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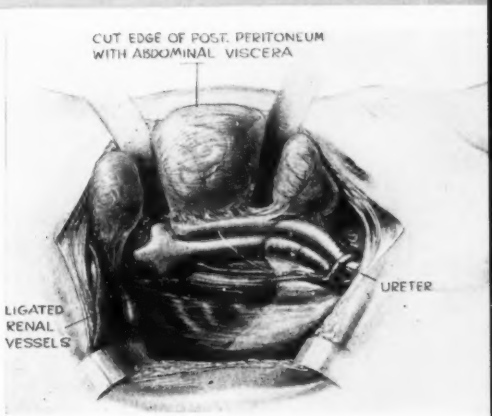

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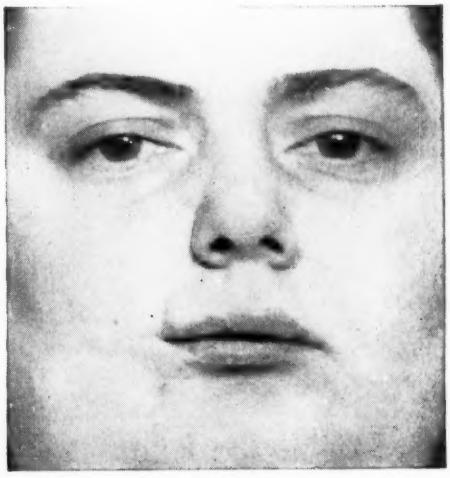

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A.M.A. Archives OF SURGERY

Editorials

SURGICAL ASPECTS OF SPLENIC DISEASE

ALTHOUGH the spleen is an important component of the reticuloendothelial system, its removal may be beneficial or curative in many hematologic diseases. With the advent of sternal aspiration, which is a relatively simple technique, and with better knowledge of the functions of the hematopoietic system, more and more indications for splenectomy have been found. There are still many gaps in knowledge of splenic physiology, particularly with regard to its effect on bone marrow. The excellent article, "The Surgical Aspects of Splenic Disease," by Cole, Majarakis, and Limarzi, appearing in this issue, summarizes the known facts and presents a concise discussion of the theoretic aspects of splenic function. The indications for splenectomy given by these authors are sound, and their results are in accord with the experience of my colleagues and me.

As set forth in this article, there are two conditions for which splenectomy may be curative, namely, thrombocytopenic purpura of the essential type and familial hemolytic icterus. The latter condition is almost invariably cured by splenectomy, whereas this procedure produces cure in approximately 85% of patients who have so-called idiopathic thrombocytopenic purpura. As the authors bring out, it must be admitted that results of splenectomy in most other situations are unpredictable and the indications for the operation cannot be rigidly defined. It is indeed discouraging to observe that the condition in certain cases is cured by splenectomy, while in other cases, apparently similar in all aspects, no benefit is derived.

A tendency exists at present to regard most of the situations in which splenectomy is indicated as instances of hypersplenism. It is well known that in some way the normal spleen has an inhibitory effect on the maturation and release of the various elements of the bone marrow, as well as a certain destructive activity, and that its removal is followed by at least a transient increase in the number of circulating erythrocytes and platelets. Accentuation of this normal function has been termed "hypersplenism," which manifests itself by inhibition of hematopoiesis or by blocking the release of mature cells from the bone marrow. The spleen is definitely enlarged in almost all instances.

One may set forth certain general ground rules for hypersplenic syndromes as follows: (1) the presence of splenomegaly, with the exception of essential thrombocytopenic purpura; (2) a reduction in all, or a selective reduction in some, of the formed elements of the peripheral blood, and (3) the presence of normal or hyperplastic bone marrow with (as in the case of essential thrombocytopenic purpura) arrest of maturation of certain of the cellular elements. Particular care must be taken to rule out leukemias, particularly of the aleukemic type; lymphomas; myeloid metaplasia of the spleen associated with myelofibrosis; erythroblastic leukemias, and

certain splenomegalies such as might occur in typhoid fever and malaria; in collagen diseases such as lupus erythematosus, amyloidosis, and Felty's syndrome, and in storage diseases such as Gaucher's disease and Niemann-Pick disease.

With the introduction of therapy with steroid agents, it is possible in certain conditions to use these substances prior to consideration of splenectomy. This may be particularly true in instances of acquired hemolytic anemia, when, if at all possible, the patient should have a trial of either corticotropin (ACTH) or cortisone before splenectomy is recommended. In certain severe hemolytic anemias and in certain thrombocytopenic purpuras, corticotropin given by intravenous drip may be life-saving. If steroid therapy fails to modify the process favorably, splenectomy should be recommended, although no promise can be made that any benefit will result.

Until greater knowledge of the relationships between the spleen and other hematopoietic organs is available, the best criteria at present to provide an indication for splenectomy are the following: (1) normal or hyperfunctioning bone marrow with or without evidence of arrest of maturation of certain formed elements (sternal aspiration usually reveals this, but more than one specimen of marrow may be required to obtain material that is truly representative of the condition of the entire marrow, and occasionally it may be necessary to perform trephination to obtain a button of marrow for biopsy); (2) enlargement of the spleen, and (3) reduction of formed elements in the peripheral blood, either in their entirety or in certain selective cellular groups.

Care must be taken to differentiate those proliferative diseases associated with splenomegaly, particularly the lymphomas and leukemias, although in certain instances a hemolytic process may be associated with chronic lymphatic leukemia and certain of the lymphomas. In such instances, improvement in the hemolytic process may be obtained by splenectomy but the basic disease continues unaltered.

CHARLES H. WATKINS, M.D.
Section of Medicine, Mayo Clinic.

PROPHYLACTIC ANTIBIOTIC THERAPY

SOME OF THE current practices of antibiotic prophylaxis are becoming matters of considerable concern. Antibiotic therapy has unquestionably had a profound effect on the practice of surgery, but the persistent trend toward extending antibiotic prophylaxis indiscriminately to every patient undergoing an operative procedure is creating a number of serious problems.

In the first place, it has produced in many surgeons a false sense of security which is not based upon fact. The use of antibiotics may give an erroneous impression that everything possible has been done for the prevention of an infection and that any infection developing in the wound does so because of the failure of the antibacterial action of the agent used rather than the neglect of established surgical principles or important technical details. The occurrence of the infection is usually the result of inadequate removal of devitalized tissue and foreign bodies or the development of devitalized tissue postoperatively as the result of ill-fitting dressings or casts, strangulation of large bits of tissue, and impaired circulation secondary to delayed thrombosis or tight closure of wounds. Experience empha-

Recorded for publication March 29, 1955.

From the Department of Surgery of the College of Medicine, University of Cincinnati, and the Cincinnati General Hospital.

sizes that antibiotics are most effective prophylactically when used as adjuvants to careful and adequate surgery but not as substitutes for it.

In addition, infections developing during prophylactic therapy may be so masked that the surgeon is unaware of their existence because of the absence of fever or local signs of inflammation. In reality, however, they may be dissecting, extensive, and even lethal. In other instances, fever may occur, but the absence of the expected local signs of infection may lead the surgeon to assume incorrectly that the fever has a different cause.

Other evidence of this blind reliance on antibiotics is a tendency to disregard the significance of white blood cell counts and differential counts during the postoperative state. Surgeons and residents have at times failed to obtain such cell counts, both in civilian hospitals and in military evacuation hospitals, under the belief that infections will not occur under prophylactic antibacterial treatment. It has become more and more difficult to convince interns that the information yielded by white blood cell counts and differential counts justifies the labor and time necessary to do them.

Two other effects of widespread therapy for trivial wounds or minor conditions are sensitization of an increasing proportion of the population to the different agents and a progressive acquisition of resistance by bacteria in our environment to the various antibiotics. In the case of penicillin, the number of patients who have acquired sensitivity is considerable. If the treatment which produced this state was in fact unnecessary, it is unfortunate that these persons should be denied the further benefits of this agent or be subjected to serious risks if it is given without good reason.

Acquired resistance of bacteria in our environment to the antibacterial agents is a more serious effect on the population as a whole. An important example of this has been the observation throughout the United States of progressive resistance of the pathogenic staphylococci first to penicillin and, more recently, to other chemotherapeutic agents. This has been particularly true in hospital practice. It is generally agreed that the majority of strains of staphylococci are now resistant to penicillin, and many are resistant to chlortetracycline (Aureomycin), oxytetracycline (Terramycin), and streptomycin. All but a few, however, are still sensitive to erythromycin, chloramphenicol, and bacitracin. The persistence of a high percentage of staphylococcal susceptibility to chloramphenicol (Chloromycetin) has been of particular interest. The factors responsible for this are not clear.

In 1943, at the time of the introduction of penicillin for clinical trial, 93%-95% of the virulent staphylococci were quite sensitive to penicillin. During the ensuing 12 years this percentage has gradually decreased year by year to levels varying from 12% to 57% in different localities and in different reports. In addition, there is evidence that the assets of the newer antibiotics are being slowly dissipated by similar changes. With some of the bacteria such as the beta-hemolytic streptococci and pneumococci, this has not been true, but with many others, particularly the Gram-negative bacilli of the colon group, acquired resistance to the broad-spectrum agents has been and is an important factor. Coliform bacteria have been observed to undergo a 50,000-fold increase in resistance after only 48 hours of exposure to streptomycin. Similarly, many of the patients in the tuberculosis sanitoria today harbor streptomycin-resistant tubercle bacilli. Time will tell whether the same phenomenon will be an important problem with isonicotinic acid hydrazide. Studies in our laboratory indicate that the progressive incidence of

resistance can be correlated with the amount and duration of the use of the antibiotic. Presumably, the increase in antibiotic resistance results from the suppression of sensitive bacteria and selective proliferation of resistant variants arising by mutation during therapy. There can be no doubt but that the problem is a serious one and that it will become more serious.

The not infrequent conversion through prophylactic treatment of a minor illness to a grave one because of the development of complications is also worthy of consideration. In addition to hypersensitivity states, secondary or superimposed infections may develop within a wound or elsewhere in the body which may be very serious. We do not know all the factors which contribute to the development of this complication. It is clear, however, that the infection is caused by micro-organisms other than the primary etiologic agent which are resistant to the antibiotic agent in use. Instances of spontaneous septicemia produced by strains of B. proteus have been observed by us as examples of superinfections. Other examples include pseudomembranous ulcerative enterocolitis and monilial secondary infections of the mouth, rectum, vagina, or respiratory tract. It should be emphasized, however, that the laboratory demonstration of Candida in specimens of such patients does not necessarily indicate that their presence is associated with clinical infection.

The question has been raised as to whether or not fungus infections are increasing as a result of antibiotic therapy. While this question cannot be answered definitely, there is little doubt as to the occurrence of such infections during antibiotic treatment and their difficulty in management when established. On the other hand, experimental evidence indicates that the wide-spectrum antibiotics do not enhance directly the growth of Candida albicans in vitro or potentiate mycotic disease in animals with experimental moniliasis, blastomycosis, histoplasmosia, coccidioidomycosis, and sporotrichosis.

The toxicity of the various antibiotics themselves differs considerably, but most if not all of them have been shown to be capable of producing one or more types of reaction. Those caused by overdosage can be readily prevented or controlled. Those secondary to an idiosyncrasy may be very serious. Idiosyncrasy to chloramphenicol with depression of the bone marrow and leucopenia, agranulocytosis, or aplastic anemia received considerable publicity in 1951 and 1952. During the past four years approximately 75 proved cases of blood dyscrasia have been collected from the literature. The anticipated incidence of this complication has been fixed at 1 in 40,000.

It is a common practice to give penicillin before and after operations to patients undergoing elective "clean" surgical procedures for the dual purpose of protecting the operative site against infection from accidental contamination and of preventing pulmonary complications. It may fail in both purposes. When it does, the infection developing is resistant to penicillin and usually much more difficult to treat. For most elective operations in clean, healthy tissues, there is no need for such prophylaxis except possibly after those of the central nervous system or thorax. It is generally preferable to withhold all such treatment until signs of incipient infection appear, since many of them are more likely to be controllable by penicillin.

With the extraordinary advances resulting from antibiotic therapy in mind, one should not make the serious error of forgetting other forms of treatment.

The above considerations emphasize the necessity of determining the specific indications for prophylactic antibiotic therapy on the basis of our existing knowledge in an effort to minimize the undesirable effects under discussion.

In civilian surgical practice, the existing evidence indicates that the prophylactic use of antibacterial agents is of value under the following conditions:

1. In contaminated wounds of violence, including burns.

2. In elective surgical procedures performed through, or in, contaminated areas such as the gastrointestinal, respiratory, or genitourinary tracts.

3. In patients with associated derangements of the urinary tract who undergo surgical operations.

4. In patients with indwelling catheters.

5. In persons with preexisting valvular heart disease who sustain injuries or undergo operations of the oral or pharyngeal cavities.

6. In patients undergoing emergency surgery in the presence of associated but unrelated infections such as acute tonsillitis.

7. For preparation of the gastrointestinal tract.

8. For elderly people with chronic pulmonary disease who undergo surgical treatment.

The prophylactic use of antibacterial agents for the prevention or suppression of infection developing in contaminated wounds is worthy of special consideration. Their chief value lies in the attenuation, limitation, or control of infection by residual bacteria in wounds after débridement. Another asset is the localization of infection developing within wounds of patients in whom surgical treatment is necessarily delayed or impossible. In both civilian and military practice the use of these agents has unquestionably produced significant results in the control of wound infections and reduction of mortality. However, infections complicating wounds of violence still remain a very important problem, antibiotic therapy notwithstanding.

Subacute bacterial endocarditis develops in patients with congenital heart disease and in persons who have had rheumatic fever with cardiac involvement. Operative procedures in the mouth, including tonsillectomy and tooth extraction, may initiate this infection by a viridans Streptococcus. Whenever such surgical procedures are contemplated for a patient with one of the above-mentioned heart conditions, it is advisable to administer penicillin or sulfadiazine prior to and subsequent to surgery. The cooperation of physician and surgeon is helpful in preventing this dreaded complication.

A motivating factor in the surgeon's indiscriminate use of antibiotics unquestionably is the request or demand of the patient or his family. Too often these agents are given to bolster the confidence of the patient, his relatives, or his family physician. This is not a justifiable excuse, however, and active and persistent education of the public as well as the medical profession is necessary to eliminate this source of pressure.

Another factor demanding a critical review of the practice of prophylactic antibiotic therapy is an economic one. There is little doubt that the present practices are wasteful and that they cause expense to patients, insurance companies, and hospital administrators. We have been unable to obtain accurate figures from the isurance companies, but at the Cincinnati General Hospital, a university teaching

hospital, the expenditure for antibiotics has steadily increased from $24,467.64 in 1950 to $35,764.27 for the first 10 months of 1954.

It is important for every surgeon to know the indications for intelligent antibiotic therapy and to be familiar with its limitations, dangers, and consequences.

How can these aims be accomplished? More thoughtful consideration of the indications of antibiotic therapy by all physicians on the basis of the individual requirements of cases would do much to achieve these ends. In addition, active education of the general population is suggested to acquaint them with the dangers of the indiscriminate use of antibiotics and to eliminate their expectation of antibiotic therapy in the absence of definite indications as determined by the physician. This could be accomplished through the cooperative efforts of the newspapers and local medical societies.

WILLIAM A. ALTEMEIER, M.D.
WILLIAM R. CULBERTSON, M.D.
and
MARK VETTO, M.D.

The Fractured Rib—A Significant Injury

An Analysis of Seven Hundred Thirty Consecutive Cases

RICHARD L. RAPPORT, M.D.
ROBERT B. ALLEN, M.D.
and
GEORGE J. CURRY, M.D., Flint, Mich.

The fractured rib is often an injury of considerable consequence. Yet it is frequently neglected, especially when associated with other major trauma. More than 5% of the patients admitted to the section for the surgery of trauma at Hurley Hospital during the six-year period of this report presented such an injury. Textbooks give little consideration to this subject and imply that it is not significant. Christopher states: "All fractures of the ribs save compound ones and those accompanied by severe crushing injury of the chest and its contents may be considered as minor." [1] As recently as 1940 Hinton and Steiner stated, "The paucity of the literature on fractures of the ribs in the past ten years is readily explained by the frequency of the fracture and the relative infrequency of complications." [2]

Recorded for publication Jan. 24, 1955.

From the Section for the Surgery of Trauma (Division of Thoracic Surgery), Hurley Hospital, Flint, Mich.

Read at the Eleventh Annual Meeting of the Central Surgical Association, Detroit, Feb. 19, 1954.

TABLE 1.—*Review of the Literature: Complications and Mortality of Fractured Ribs*

Author	Year	Cases, No.	Complications, Per Cent	Mortality, Per Cent
Ziegler [3]	1910	820	7.8	0.85
Haubensek [4]	1923	1,156	13.0	1.81
Bistolfi [5]	1930	572	11.2	....
Findlay [6]	1937	163	...	21.5
Hinton & Steiner [2]	1940	279	29.7	10.4
Richardson & Papper [7]	1947	217	33.3+	4.1
Cameron et al.[8 *]	1949	109	65.0	10.0
Heroy & Eggleston [9]	1951	492	35.0	10.7

* Multiple rib fractures only (three or more ribs).

TABLE 2.—*Thoracic Trauma (1947-1952)*

Total Thoracic Injuries 832

Injury	No.
Closed	
With fracture	715
Without fracture	28
Total	743
Open	
With fracture	15
Without fracture	74
Total	89

Because of the proximity of the bony rib cage to vital underlying organs complications tend to be serious. The fractured rib frequently is an extensive injury responsible for a formidable list of complications and is associated with a relatively high mortality rate. This is substantiated by a review of the literature (Table 1).

MATERIAL

During the years 1947 to 1952, inclusive, 730 patients with rib fractures were seen in Hurley Hospital. There were 490 inpatients (67%) and 240 outpatients (33%).

During this same period a total of 832 thoracic injuries were seen (Table 2). Seven hundred forty-three were closed injuries, only 28 (4%) of which were not associated with rib fractures. On the other hand, 74 (83%) of 89 patients with open injuries did not have fractured ribs.

Associated Injuries.—Two hundred fifty-eight patients (52.6% of those admitted to the hospital) suffered a total of 395 associated injuries (Table 3). Most are significant as regards morbidity and mortality. Special mention should be made of cranio-

TABLE 3.—*Associated Injuries*

Injury	490 Inpatients No.
Craniocerebral	126
Extremity fracture	91
Fracture clavicle	43
Fracture pelvis	28
Intra-abdominal injury	28
Fracture scapula	20
Fracture vertebral body	19
Fracture transverse process	18
Cervical	3
Dorsal	5
Lumbar	10
Fracture sternum	15
Ruptured diaphragm	7
Total (injuries in 258 patients)	395

cerebral injuries, since coma, with resultant loss of cough reflex leading to retained secretions, atelectasis, and pneumonitis tends to complicate the appraisal of a primary thoracic injury. Many associated injuries restrict the examiner in his evaluation of the chest because of limitations in positioning the patient. As in all patients with trauma, the individual must be considered as a whole and no one injury neglected for another.

COMPLICATIONS AND SEQUELAE

Of the 490 patients admitted to the hospital 41% presented some complication associated with the fractured rib. We have divided complications into five anatomical groups: (1) chest wall, (2) pleural, (3) pulmonary, (4) mediastinal, and (5) abdominal (Table 4). There were a total of 444 complications in 202 of the 490 patients admitted to the hospital. Thus, complications occurred in 41% of the hospital patients, or in 28% of the entire group.

Chest Wall (Table 5): All fractured ribs are associated with hemorrhage in the chest wall; hence this has not been considered a complication. Intercostal neuralgia, osteomyelitis, and intercostal aneurysms were not found.

TABLE 4.—*Complications*

	202 of 490 Inpatients (41%) No.	Per Cent
Chest wall	88	17.9
Pleural	130	26.5
Pulmonary	160	32.6
Mediastinal	8	1.6
Abdominal	58	11.8
Total	444	

Subcutaneous emphysema, reported in 67 patients, was the most common complication. This appears alarming when extensive, but is seldom serious * and specific treatment is unnecessary since a patent airway is always present.[11]

Paradoxical respirations occur as a result of multiple rib fractures with a flail or "stove-in" chest. Twenty-one of our patients had such an injury, and 7 of the 34 patients who died presented this complication. Immediate fixation is required.

TABLE 5.—*Complications—Inpatients*

1. Chest Wall	No.	Per Cent
Subcutaneous emphysema	67	13.7
Paradoxical respirations	21	4.2
Total	88	17.9

TABLE 6.—*Complications—Inpatients*

2. Pleural	No.	Per Cent
Hemothorax	62	12.7
Pneumothorax	59	12.0
Pleurisy with effusion	9	1.8
Total	130	26.5

We have used the simpler methods of positioning, strapping, and sand-bagging as emergency treatment. If these prove inadequate, skeletal traction or internal fixation may be necessary.

Tracheotomy is frequently done to reduce the dead air space, to decrease resistance to respiration, and to provide easy access for removal of secretions.

Pleural Complications (Table 6): These complications include hemothorax, pneumothorax, pleurisy with effusions, and chylothorax. One hundred thirty of our cases had such complications. Others have found significant incidence of this complication.†

Hemothorax: Sixty-two of our cases had blood in the pleural space. Hemorrhage may

* References 8 and 10.

† References 8, 10, 12, 13, and 14.

occur from parenchymal, intercostal, internal mammary, or great vessels.

Prior to World War II opinion was divided as to the ideal treatment for hemothorax. Recently, however, it is agreed that early and complete aspiration should be accomplished.‡ Thoracentesis should be done "early, often, and diligently and without air replacement" to "reduce the incidence of chronic clotted hemothorax, empyema, and atelectasis, resulting in pulmonary suppuration, altered pulmonary function, and permanent disability."[16] Enzymatic débridement has been used to liquify the clot, but we agree with Valle[14] that resultant pain, febrile reactions, and failure to show improvement by x-ray make this procedure of doubtful value. If hemorrhage is progressive, despite thoracentesis, early thoracotomy with evacuation of clots and control of hemorrhage is indicated. Indeed, McEachern employs this as a primary procedure if over one-third of the lung is encased by hemothorax.[24] Organizing hemothorax may occur despite treatment. If loculation should prevent effective thoracentesis or if fibrothorax results, decortication is performed between the third and the fifth week.§

Pneumothorax: Air in the pleural space was demonstrated by roentgenography in 59 patients (Table 6). Pneumothorax may result from either open or closed injuries.

Open wounds with chest wall defects ("sucking wounds") should be converted immediately into closed wounds by occlusive pressure dressings and the pneumothorax then treated appropriately.

There is general agreement as to the management of tension pneumothorax which may constitute a true surgical emergency. Since the signs and symptoms are quite characteristic, there is no need (and often no time) to await roentgenologic diagnosis. Air should be withdrawn by thoracentesis at once.

Small, uncomplicated, closed pneumothoraces likewise present no controversy. If the collapse is less than 20 to 25%, no specific treatment is required. It should be emphasized, however, that the degree of collapse is often underestimated on the roentgenogram. Moreover, what initially is an insignificant amount of collapse may progress so that repeated observations are essential.

Conflicting viewpoints are held concerning the management of closed pneumothorax with more than 25% collapse. Some believe active intervention is not indicated unless there are signs of excessive tension.‖

One of us (R. L. R.) recently discussed the management of spontaneous pneumothorax and concluded that closed catheter drainage should be instituted if collapse is greater than 25%.[27] This applies equally to traumatic pneumothorax. Indwelling catheter with water-seal drainage has been recommended by many.¶

The advantages of closed catheter drainage are manifold. Complete early reexpansion of the lung is desirable for prevention of pleural effusion, persistent bronchopleural fistula, and empyema. Most effective closure of the parenchymal tear is by reexpansion of the lung so that visceral and parietal pleura come into contact, with sealing of the fistula by adhesion of the pleural surfaces at the site of rupture.

Pleural effusion is an infrequent sequela. It was seen in nine of our patients. Effusion seldom occurs in the presence of hemothorax or pneumothorax if early and vigorous efforts are made to reexpand the lung. Management should consist of persistent thoracentesis.

Chylothorax is a rare complication and was not seen in our series. Baldridge and Lewis[29] collected 59 cases in 1947.

Pulmonary Complications (Table 7): Pulmonary complications occurred most commonly, comprising 32.6% of the 202 cases of inpatients with complications. These are interrelated, and two or more frequently were present in the same patient. The term "traumatic wet lung" has been given to various combinations of these complica-

‡ References 8, 9, 12, 13, 14, 15, 16, 17, 18, 19, and 20.

§ References 12 and 14.

‖ References 2, 6, 25, and 26.

¶ References 8, 10, 14, 15, 18, 19, 20, 23, and 28.

tions.# We approve of this terminology since it implies the sequence of events: retained secretions, tracheobronchitis atelectasis, and pneumonia.

The management of these complications is extremely important and along with pneumothorax and hemothorax constitute the triad that must be looked for at frequent intervals and when discovered treated vigorously.

Adhesive strapping and administration of opiates are mentioned only to be condemned. The first consideration must be for an unobstructed airway and evacuation of the tracheobronchial tree. Intercostal nerve block, done either in the intercostal space or paravertebrally, is most important and should be done early. When pain is relieved, the patient coughs and clears his own secretions dramatically. If cough is impossible or ineffective, nasotracheal catheter aspiration or bronchoscopy may be necessary. Samson and Brewer have given an excellent discussion of the indications for each.[31] Tracheotomy may be advisable,* and we are doing this more and more frequently, with gratifying results.

TABLE 7.—*Complications—Inpatients*

3. Pulmonary	No.	Per Cent
Contusion	62	12.6
Tracheobronchitis	49	10.0
Pneumonia	29	5.9
Atelectasis	18	3.7
Pulmonary edema	1	0.2
Pulmonary abscess	1	0.2
Total	160	32.6

Pulmonary abscess occurred only once. However, this danger is ever present, considering the potential for infection provided by areas of hemorrhage into pulmonary parenchyma together with the ready source of infection from traumatized and contaminated bronchioles with bronchopleural fistulae. If the lung abscess fails to respond to conservative measures, early thoracotomy with decortication or resection is indicated.

References 8, 9, and 30.

* References 20 and 32.

TABLE 8.—*Complications—Inpatients*

4. Mediastinal	No.	Per Cent
Pneumomediastinum	2	0.4
Hemomediastinum	1	0.2
Cardiac injuries	3	0.6
Laceration—aorta	1	0.2
Traumatic asphyxia	1	0.2
Total	8	1.6

Mediastinal Complications (Table 8): Mediastinal complications are relatively rare in the injured person who reaches a hospital.

Pneumomediastinum was observed in only three patients and was not a serious situation in any.

Cardiac injuries were diagnosed in only three patients, and injury to the great vessels was demonstrated only once. Others have reported an incidence of from 2.5 to 16%.†

Traumatic asphyxia refers to that situation where there is a dusky cyanosis of the upper part of the body following a compression injury of the chest. It results from momentary reversal of superior vena caval blood flow into its valveless tributaries, with distention and rupture of small venules and capillaries. If the patient survives the first few hours, the prognosis is good. There was one such patient in our group.

Abdominal Complications (Table 9): Certain abdominal injuries are so frequently related to thoracic trauma that we consider them complications rather than associated injuries. In all our cases the rib fracture

† References 6, 9, 33, 34, 35, 36.

TABLE 9.—*Complications—Inpatients*

5. Abdominal	No.	Per Cent
Ruptured abdominal organs	28	5.7
Kidney	9	
Liver	9	
Spleen	6	
Small intestine	2	
Stomach	1	
Adrenal	1	
Ileus	23	4.7
Lacerated diaphragm	7	1.4
Total	58	11.8

was directly related to the abdominal complication.

Ruptured Abdominal Viscera: Fifty-eight patients developed pathology at or immediately below the diaphragm. Trauma to abdominal organs was found in 28 cases, or 5.7% of all complicated cases of rib fractures. The kidney and liver were most frequently involved and were closely followed by the spleen. Details of management of these problems are not in the realm of this discussion. The problem is amplified when one considers how often signs and symptoms of chest injury are referred to the abdomen in the absence of intra-abdominal pathology. The differential diagnosis between true abdominal trauma and referred pain is often difficult and requires frequent examinations, including evaluation of the abdomen after appropriate blocks of intercostal nerves.

Lacerated diaphragm was found in seven patients. Some degree of pleural effusion and hemothorax was always present; consequently the diagnosis was sometimes difficult to establish by both physical examination and standard roentgenograms. Surgical exploration should be done early in all patients whose condition permits. Delayed repair may be done in severely injured patients who survive.

Ileus was present in 23 cases (4.7%) in which there were no intra-abdominal injuries or fractures of the spine or pelvis. Treatment is by intestinal intubation and careful observation for other causes which may require active intervention.

MORBIDITY AND MORTALITY

The average hospital stay for patients with uncomplicated rib fractures without associated injuries was 4.8 days. Those patients with complications associated with the fracture but without other injuries were hospitalized for an average of 16.4 days.

Autopsy was performed in 27 (79%) of the 34 patients who died (Table 10). It is sometimes difficult, even with autopsy, to evaluate the role thoracic injury played in these deaths. Ten of the 34 patients who died had thoracic injuries too minor to have contributed to death. Seven of these were due to craniocerebral injuries and the remaining three to associated medical problems. A second group, of 11 patients, had associated injuries lacking or insignificant, so that death was attributed to the thoracic injury. In a third group, of 13 patients, serious injuries of the thorax existed concomitant with other associated injuries, and so the cause of death could not be established. In 24 patients the thoracic injury was either the sole or a decisively contributing factor to death. Thus, we have established a mortality rate of 4.9% for the 490 patients admitted to the hospital, or 3.3% for the total group of 730. This compares with a mortality rate variously reported ranging from 4.1 to 24.5%.‡

TABLE 10.—*Mortality*

Due to thoracic injury	12
Due to associated injury	10
Due to both	12
Total	34

Thoracic injury contributed to death in 24 patients: 4.9% of 490 patients admitted to the hospital; 3.3% of 730 patients with fractured ribs.

Table 11 lists the frequency of complications in those in whom thoracic injury contributed to death. Pulmonary contusion or laceration occurred in 13. In no instance was death attributed to the contusion or laceration per se, but other complications were always present.

Traumatic wet lung was likewise present 13 times in those who died. While other major complications were often associated,

‡ References 2, 6, 7, 8, 9, and 37.

TABLE 11.—*Complications in Relation to Mortality*

Complication	No.
Pulmonary contusion and/or laceration	13
Traumatic wet lung	13
Hemothorax	11
Pneumothorax	10
Ruptured viscera	10
Paradoxical respirations	6
Subcutaneous emphysema	4
Ruptured diaphragm	3
Ileus	2
Cardiac injury	1
Hemomediastinum	1
Pleural effusion	1
Ruptured aorta	1

this frequently seemed to be the true cause of death.

Hemothorax was noted in 11 of the patients who died. Active evacuation of the blood by the best available method has resulted in a decreased mortality from this cause.

Pneumothorax was present in 10 of the patients who died. It was the immediate cause of death in one patient, an 83-year-old man who died four hours post-trauma with no other significant injury. His pneumothorax was untreated. If adequately treated by closed thoracotomy tube with water seal drainage, no patient, we believe, should die from this complication.

Seven patients had paradoxical respirations. All had other complications, and we are unable to list this as the sole cause of death in any.

Ruptured diaphragm was present in three patients who died. None were operated on. All three had severe, extensive injuries, and, while the lacerated diaphragm contributed to death, it can be listed as the cause of death in none.

Six patients who died had ruptured abdominal viscera. For a variety of reasons none of these patients were operated upon.

Other complications contributing to death occurred in isolated instances. Hemomediastinum was found concomitant with a severe pulmonary contusion in a patient with multiple fractures of the extremities who expired four hours post-trauma. Pleural effusion was a late sequela in a 52-year-old patient with other pulmonary complications in addition to a craniocerebral injury. There was one cardiac injury, myocardial contusion with hemopericardium in a patient who had multiple pulmonary complications and a severe craniocerebral injury. One patient with a lacerated aorta lived long enough to be included, only because she jumped from the hospital window and was seen by a house physician immediately before death.

SUMMARY AND CONCLUSIONS

We have presented 730 consecutive cases of fractured ribs. Complications related to the fractures occurred in 41% of hospital patients (28% of the entire group). These are reviewed and prophylaxis and management briefly discussed. Of the 490 patients admitted to the hospital 4.9% died with the thoracic injury as the sole or a decisively contributing factor to death.

We conclude that the fractured rib is frequently associated with other chest injuries which are responsible for a formidable list of complications and is associated with a relatively high mortality rate.

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Blood Values in Gastric Carcinoma

Preoperative and Postoperative Blood Volume, Total Circulating Hemoglobin, and Serum Protein in Patients with Gastric Carcinoma

BJARNE FRETHEIM, M.D., Oslo

As a matter of principle, a patient who is subjected to an operation should have normal values for blood volume, hemoglobin, and serum protein and, as far as possible, should be kept in such balance during and after the operation, compensation being made for the lost blood by blood transfusions. Endeavors have been made to achieve this by preoperative control of the hemoglobin percentage, the serum protein concentration, and the fluid balance and, possibly, by supplying blood by transfusion.

The loss of blood is measured during the operation, and as much blood is supplied as the amount lost according to the rule of Wangensteen, which is the weighed loss of blood plus 20% to cover the amount of blood which escapes the weighing. It is endeavored to keep the patient in fluid balance postoperatively, and using estimates based on controls of hemoglobin percentage, to give the number of blood transfusions that may be required.

It is a clinical experience, however, that anemic patients need more blood in transfusion in order to reach normal hemoglobin percentage and serum protein concentration than might be expected according to the values found. It is also a clinical experience that—regardless of whether or not blood has been supplied during the operation in conformity with the Wangensteen rule—anemia always occurs after major operations.

From the University Hospital (Rikshospitalet), Department A of Surgery (Head: Prof. Leif Efskind, M.D.).

It has also been shown, by investigations made by me [1] that the postoperative fall in hemoglobin percentage and serum protein concentration does not occur simultaneously with the loss of blood, but lags, so that the lowest value is not measured until two or more days have elapsed, even when there is no clinical demonstration of hemorrhage after the operation has been finished. If, in addition, more or less uncontrollable hemorrhages occur after the operation, as is often the case from wound cavities and intestinal anastomoses, the condition becomes even more complicated, and it is not even possible fairly accurately to supply the correct amount of blood gradually as blood is lost, thereby giving the patients the optimal chances of being cured.

In order to approach closer to this goal, the blood volume, the total circulating hemoglobin, and the serum protein have to be ascertained, and the fluctuation of these values has to be followed preoperatively and postoperatively. I have done this in a group of patients with gastric ulcer or gastric carcinoma in which gastric resection or total gastrectomy has been carried out. Parts of these investigations have been published previously.*

The total hemoglobin and the blood volume have been determined by the Sjöstrand CO method,† which, to the best of my knowledge, is the sole method whereby daily examinations may be performed preoperatively and postoperatively with adequate precision and without harming the patient. The hemoglobin percentage has been determined as muriatic hematin, according to the Haldane standard, and the serum protein concentration, by the

* References 2, 3, and 4.

† References 5 and 6.

Kjeldahl method. The red cell volume, the plasma volume, and the total circulating serum protein have been calculated after determination of hematocrit in Wintrobe's tube centrifugation for half an hour with 3000 rpm.

In 66 patients with gastric carcinoma there was as a rule a slight reduction of the blood volume in comparison with healthy persons, and this reduction is due to a fall in red cell volume, while the plasma has not been reduced and is even higher than normal in cases with pronounced anemia. As a rule, the total hemoglobin is considerably reduced, being 23% lower than normal. The serum protein concentration is reduced just like the hemoglobin percentage. As the plasma volume is not reduced, however, or has even become higher in some cases, the total circulating serum protein is slightly reduced. Nineteen patients with gastric carcinoma received from 1 to 10 blood transfusions of 450 ml. each preoperatively, and the effect of these transfusions has been compiled in Figure 1.

After transfusion of about 2070 ml. of freshly drawn blood, the hemoglobin percentage, the total hemoglobin, and the red cell volume rise considerably towards ideal values, but no more than 70% of what might be expected according to the amount of blood supplied. In the 9 patients with the highest degree of anemia, entered in the second column on the Figure, the increase is relatively lower than in the 10 less anemic patients entered in the third column on the Figure. Evidently, as hemolysis takes place of some of the blood supplied, relatively more blood is required to bring the values up toward normal level.

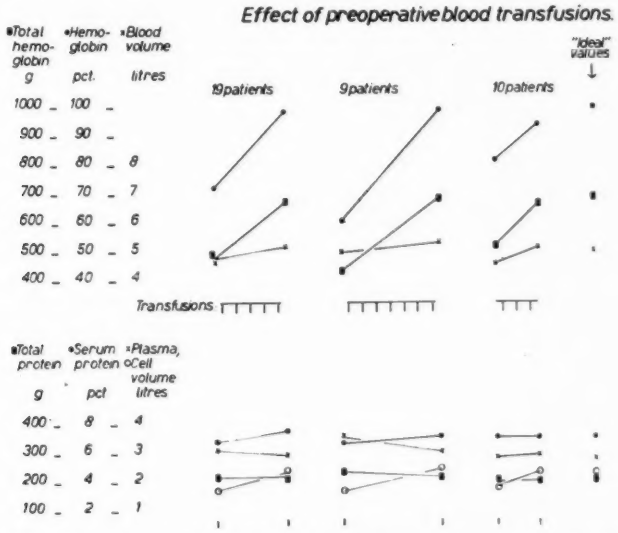

Fig. 1.—Effect of preoperative blood transfusions on hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, in 19 cases of gastric carcinoma. The calculated "ideal values" are entered on the right.

Concurrently there is a rise in the serum protein concentration but a fall in the plasma volume, the total circulating serum protein thus remaining unchanged. In the nine patients with the highest degree of anemia, the rise in serum protein concentration is less and the fall in plasma volume more pronounced, so that there is generally a fall in the total circulating serum protein, while these changes are less marked in the 10 less anemic patients.

The total result of these changes is a slight increase of the blood volume, and the result of the blood transfusions is that all values approach the ideal values. So far as the factors examined are concerned, the patients have thereby approached optimal preoperative condition.

In order to investigate the "standard reaction" to an uncomplicated gastric resection of hematologically healthy persons, a group of patients with gastroduodenal ulcer has been examined preoperatively and postoperatively. In six patients the operation could be characterized as perfectly uncomplicated; no blood transfusion was given either preoperatively or postoperatively, but fluid balance was maintained. The loss of blood has been measured by the weighing of compress blood. A graphic illustration of the average values is given in Figure 2.

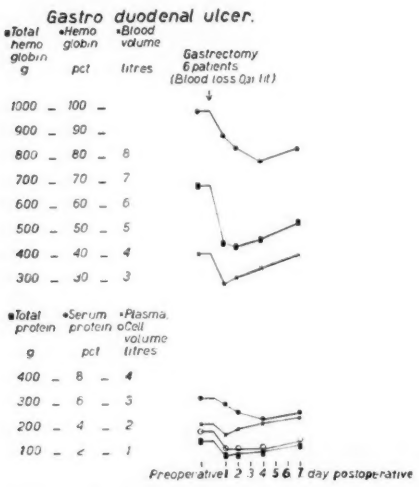

Fig. 2.—Preoperative and postoperative values of hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, in six cases of uncomplicated gastric resection for ulcer ("standard reaction").

The hemoglobin percentage falls gradually to the lowest value on the fourth day postoperatively, when it is 82% of the preoperative value, and then it rises slowly.

The total hemoglobin falls to the lowest value 24 hours postoperatively, when it is 64% of the preoperative value, remains unchanged for the next 24 hours, and then rises again with increasing rapidity to the seventh day postoperatively, when it is 80% of the preoperative value.

The blood volume also falls to the lowest value 24 hours postoperatively, when it is 77% of the preoperative value. It then rises again more rapidly than the total hemoglobin, most in the period from 24 to 48 hours postoperatively, and on the seventh day the preoperative value has been regained.

The red cell volume falls to the lowest value 24 hours after the operation, when it is 69% of the preoperative value, remaining unchanged to the fourth day postoperatively, whereupon it again rises slowly.

The plasma volume falls to the lowest value 24 hours postoperatively, when it is 81% of the preoperative value. Then it rises more rapidly than the other factors, reaches preoperative value on the third day postoperatively, and continues rising to the seventh day, when it is 11% higher than the preoperative value.

The serum protein concentration falls gradually, in the same manner as the hemoglobin percentage, to the lowest value on the fourth day postoperatively, when it is 79% of the preoperative value, and thereupon it rises slowly.

The total circulating serum protein falls to the lowest value 24 hours postoperatively, when it is 72% of the preoperative value; then it again rises slowly to the seventh day postoperatively, when it is 88% of the preoperative value.

The postoperative fall is greatest, therefore, for total hemoglobin and red cell volume and lowest for the circulating serum protein and plasma volume. The resultant figure is the fall in the blood volume, which percentually takes a medium position and which is compensated during the following days, to reach normal value again on the seventh day after the operation. Calculated by the fall in the hemoglobin amount, the loss of blood during the operation and the first 24 hours afterwards is 30% of the total amount of

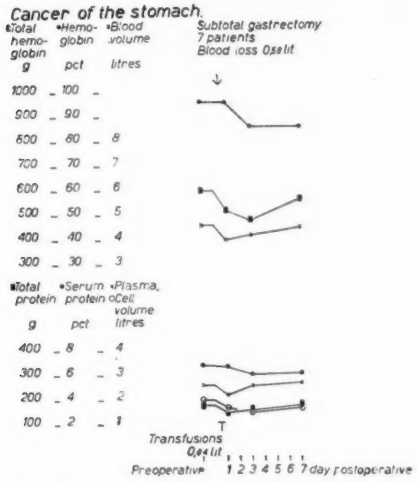

Fig. 3.—Preoperative and postoperative values of hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, in seven cases of subtotal gastrectomy for carcinoma, in which an average of 260 ml. more blood was given in transfusion than the weighed loss of blood during the operation.

blood, while the loss of blood weighed during the operation is 6% of the amount of blood.

Seven patients in whom subtotal gastrectomy has been performed for carcinoma have been examined by the same procedure. Even if conditions here are considerably more complicated, partly because some of the patients received preoperative blood transfusions, partly because the patients received a varying number of blood transfusions during the operation, and partly owing to greater variation in surgical procedures, the course of the curves preoperatively and postoperatively shows great similarity to the "standard curves" (Figure 3).

The essential difference from the "standard curves" is that the total hemoglobin and the red cell volume continue falling to the third day postoperatively before they again commence rising. This occurs in spite of 260 ml. more blood having been given in transfusion than the loss of blood weighed and is supposed to be due to destruction of some of the red blood cells supplied, as there was no sign of persisting hemorrhage 24 hours postoperatively.

More considerable changes are found from the "standard reaction" in cases of abdominothoracic total gastrectomy plus splenectomy and resection of the pancreas (Fig. 4).

In spite of the ample supply of blood, the postoperative fall in total hemoglobin, blood volume, and red cell volume is greater than in subtotal gastrectomy; otherwise, it shows a similar course.

The plasma volume, on the other hand, shows a different relation, having fallen in 24 hours percentually more than the red cell volume, and remains low until the third day after the operation, when it again commences rising. The consequence is that the hemoglobin percentage rises higher postoperatively than preoperatively, giving the false impression that the loss of blood has been compensated.

The total circulating serum protein falls percentually more than in subtotal gastrectomy. Owing to the concurrent fall in plasma volume, however, the serum protein concen-

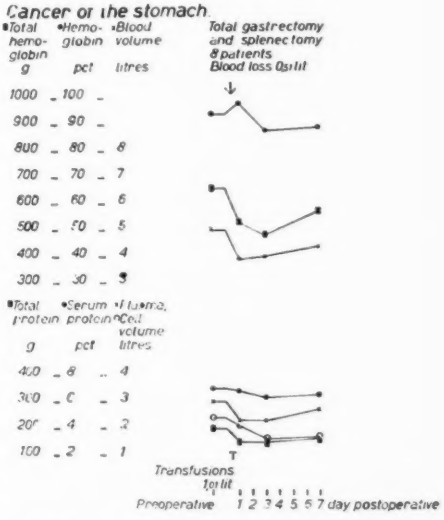

Fig. 4.—Preoperative and postoperative values of hemoglobin percentage, total hemoglobin, blood volume, and red cell volume, as well as serum protein concentration and total circulating serum protein, in eight cases of total gastrectomy plus splenectomy and resection of the pancreas, in which during the operation 500 ml. more blood was given than the weighed loss of blood.

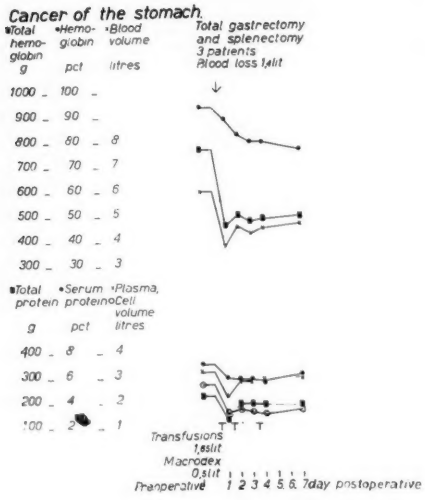

Fig. 5.—Preoperative and postoperative values of hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, in five cases of total gastrectomy plus splenectomy, in which in addition to 250 ml. more blood than the loss of blood weighed, 0.5 liter of Macrodex (Dextran) was given.

tration shows approximately the same relations as in subtotal gastrectomy, falling percentually less than in the "standard curves."

The explanation of these deviations is that the loss of blood in total gastrectomy plus splenectomy and resection of the pancreas is considerably greater than is indicated, on the basis of loss of blood weighed, and the compensation by blood transfusions is too small. A state of preshock develops, with hemoconcentration and loss of plasma-like fluid from the blood stream. The organism conquers the preshock, however; plasma-like fluid is sucked back into the circulation, and the blood volume again increases.

The excessive fall in plasma volume by preshock may be prevented by administration of dextran (Macrodex), and this also minimizes the fall in blood volume. On the other hand, it then appears as if the regeneration of hemoglobin is retarded; consequently, anemia persists for a long period (Figure 5).

In three patients on whom abdominothoracic total gastrectomy plus splenectomy had been performed, shock was imminent at the finish of the operation, and they were given 0.5 liter of dextran in addition to blood. This appears to have had the effect that the fall in plasma volume did not become as profound as in the preceding cases, and it also rose again more rapidly. This is the case also with the total circulating serum protein, and so it seems as if plasma-like fluid is sucked back into the blood serum. The total circulating hemoglobin, despite further blood transfusions, shows only a slight tendency to rise, and the hemoglobin percentage therefore continues falling. It is difficult to draw definite conclusions here, however, owing to the complicated conditions.

One patient was examined 24 hours postoperatively in a state of manifest shock

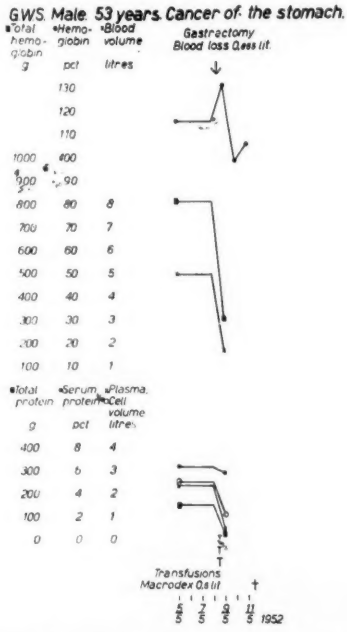

Fig. 6.—Preoperative and postoperative values of hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, in one patient in whom manifest shock developed after total gastrectomy plus splenectomy for carcinoma.

(Figure 6). Here the postoperative changes in Figure 4 are found in accentuated form. Fall in total hemoglobin, blood volume, and red cell volume is marked, the fall in plasma volume, however, and in total circulating serum protein being relatively still more pronounced. The result is a considerable hemoconcentration and a great postoperative rise in hemoglobin percentage and an insignificant fall in serum protein concentration.

During the operation, 500 ml. more blood had been given than the loss of blood weighed and, in addition, 500 ml. of dextran. The case was misinterpreted because only the result of the postoperative examination of hemoglobin percentage was available and not the result of total hemoglobin and blood volume studies. Routine fluid regimen was instituted, with the result that irreversible shock developed. Postmortem examination revealed a profuse amount of blood in the intestines and necrotic changes in the tail of the pancreas.

COMMENT AND CONCLUSIONS

Patients suffering from gastric carcinoma usually have a reduced hemoglobin percentage. Even if they have not, they are still usually anemic, their total hemoglobin being reduced. When this is not revealed in the hemoglobin percentage, it is due to a concurrent reduction of the blood volume.

The reduction of the blood volume is due in the first instance to a fall in the red cell volume, while the plasma volume is little or not at all reduced or, in some markedly anemic patients, even increased. The serum protein concentration is reduced as a rule, while the total circulating serum protein is less reduced or may even be increased when there is considerable increase of plasma volume.

The deviations from the normal are more marked the more anemic the patients are, but under all conditions it is the blood volume which shows the least deviation, as the plasma volume increases with reduced red cell volume and vice versa, the consequence being that the blood volume is kept close to normal if there is not a concurrent uncompensated loss of fluid or inadequate supply of fluid. It seems as if the hemoglobin and the plasma protein are in dynamic equilibrium and that, to a certain extent, they substitute for each other to maintain the blood volume.

It is in conformity with clinical experience that an anemic carcinoma patient has to be given considerably more blood to bring the values up to normal than would be expected according to the values found for hemoglobin percentage and serum protein concentration. This is not due solely to the anemia being greater than supposed, judged by the hemoglobin concentration, but also to the fact that a material part of the transfused red blood cells is destroyed—in the present series averaging 30% of the red blood cells administered in the course of six days. A relatively greater part of the supplied serum protein disappears from the circulation (to reduced "depots" ?). During the administration of blood a reversal of the pathological blood changes toward normal values again takes place. Even if amounts of blood are supplied in the course of a few days exceeding by far the amount required to restore normal blood volume, this volume remains approximately normal. It appears as if it is mainly the red cell volume which determines the blood volume, on condition that there is fluid balance, while plasma-like fluid oozes out or is drawn into the blood stream according to requirement.

I had a chance of observing during a lengthy period a patient with gastric carcinoma and of following the development of these changes.

This patient was admitted into our department on June 6, 1952, to be operated on for gastric carcinoma. He was in good condition, and all was well-adapted for a radical operation. The patient refused operation, however.

Half a year later he was readmitted in a considerably worse state. Adequate preoperative treatment made possible a subtotal gastrectomy.

On Figure 7 has first been entered the supposed ideal values for hemoglobin percentage (100), total hemoglobin (765 gm.), blood volume (5.55 liters), plasma volume (3 liters), red cell volume (2.55

liters), serum protein concentration (7 gm. per 100 ml. serum), and total circulating serum protein (225 gm.), in a man of the same height as the patient (173 cm.) and "ideal" weight (73 kg.).

When first admitted into hospital, the patient had a hemoglobin percentage of 78%. Total hemoglobin was 637 gm., which is 83% of the "ideal value." The blood volume was calculated at 5.9 liters, which is 6% more than the "ideal value." The plasma volume was 3.7 liters, which is 23% more than the "ideal value," and the red cell volume, 2.2 liters, which is 14% lower than the "ideal value." The serum protein concentration was 6.6 per 100 ml. serum, a little lower than the "ideal values," and the total amount of circulating serum protein, 247 gm., a little in excess of the "ideal value."

2 liters plasma, and 1.6 liter red blood cells, the development was reversed and all values again approached "ideal values." Considerably more blood was supplied than should have been considered necessary theoretically, and yet only the serum protein concentration reached the "ideal value." The hemoglobin percentage, total hemoglobin, and red cell volume had a considerable road to travel before reaching the "ideal value." The blood volume (the plasma volume particularly, however) and the total circulating serum protein still lacked some part before coming down to "ideal value." At this juncture —too early, therefore—the patient was operated on, with an uneventful course. As a consequence of the intervention, all examined factors were thrown out of balance, and on the fourth day postoperatively

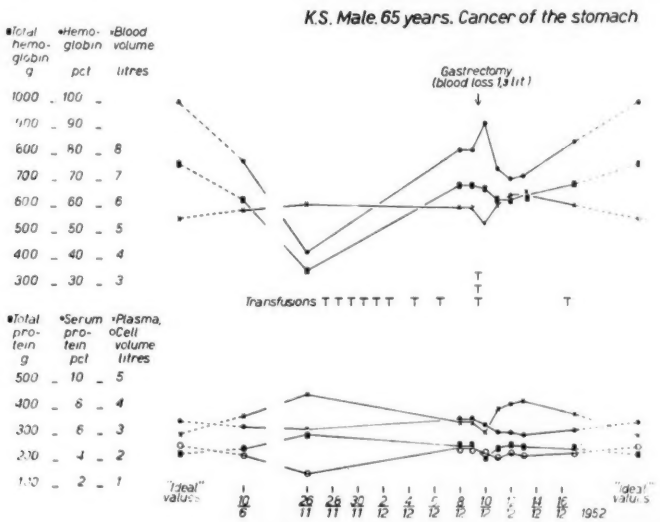

Fig. 7.—Hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as serum protein concentration and total circulating serum protein, during development of carcinoma, after treatment, preoperatively with blood transfusions, and after postoperative treatment.

Half a year later, all values had continued moving away from the "ideal." The hemoglobin was 43%, total hemoglobin, 360 gm., 47% of the "ideal value," and the blood volume had risen still more (6.05 liters). The plasma volume had increased considerably, to 4.5 liters, which is 50% more than the "ideal value," and the red cell volume had fallen materially, to 1.55 liters, which is 40% less than the "ideal value." The serum protein concentration had been reduced inappreciably, to 6.5 gm. per 100 ml., and the total circulating serum protein had risen to 295 gm. (31% more than the "ideal value").

After eight blood transfusions in the course of 10 days, of a total of 3.6 liters of blood with approximately 500 gm. hemoglobin, 140 gm. serum protein, showed the most marked changes. In a week's time, however, all values were again headed towards normal conditions.

After subtotal gastrectomy for ulcer, in which the loss of blood has not been compensated by transfusions and the patients have been subjected postoperatively to standard treatment with fluid, the hemoglobin percentage, total hemoglobin, blood volume, red cell volume, and plasma volume, as well as the serum protein concentration and the total circulating serum protein, show a postoperative fall in the various values in association

with the operation and a subsequent rise in uncomplicated cases (Figure 1). The curves for the different patients show a uniform course, permitting mean curves to be drawn, which may be designated "standard curves" for these values.

They show that the loss of blood during subtotal gastrectomy and the following 24 hours is considerably greater than the loss of blood measured by weighing of compresses and measuring of the blood in the suction flask (30% of the total amount, against the loss of blood measured as 6% of the total amount). Total hemoglobin remains almost unchanged during the three to four days following the operation and then rises again with increasing rapidity. The course of the red cell volume follows that of total hemoglobin.

The blood volume falls to the lowest value 24 hours after the operation, and then a relatively rapid increase occurs during the following days.

The plasma volume also falls to the lowest value in 24 hours, the fall here being relatively less, however, and during the following days a rapid increase takes place, so that the preoperative value has been regained on the third day postoperatively, on the seventh day being even higher than the preoperative value.

The total circulating serum protein falls to the lowest value after 24 hours, and then a regular increase occurs. On the seventh day postoperatively it is, however, still lower than preoperatively, though having increased relatively more than total hemoglobin and red cell volume.

The result of these changes is that hemoglobin percentage and serum protein concentration fall to the lowest value on the fourth day postoperatively before again commencing to increase. The hemoglobin percentage and the serum protein concentration during the two or three days following the operation are not illustrative, therefore, of the amount of blood lost and are consequently poor guides for the compensation of the blood lost, which should be instituted at the time when the loss of blood occurs, not two or three days afterward.

The fall in serum protein is relatively less than that in hemoglobin, which must be due either to the serum proteins being rapidly sucked into the blood stream from "depots" or to the fact that the production of serum protein sets in more rapidly than that of hemoglobin.

The first postoperative reaction consists in an attempt at restoring the blood volume by increase of the plasma content.

Conditions become more complicated when the starting-points are not normal. This pertains to those lesions, however, which have caused changes in hemoglobin, serum protein, and fluid balance. It also applies to cases in which infusions of blood, plasma, or dextran have been administered and, particularly, those with postoperative complications.

Still, uncomplicated subtotal gastrectomy for carcinoma (Figure 2) shows values roughly similar to the values shown after subtotal gastrectomy for ulcer.

After abdominothoracic gastrectomy plus splenectomy and resection of the pancreas (Figure 3), the actual loss of blood is still more considerable than that found by weighing, and the postoperative resection is more severe. The patients are in a state of what may be termed immanent shock, preshock, or controlled shock. It appears as if an essential change takes place in this state, viz., that plasma oozes out of the blood stream, the loss of plasma thereby becoming relatively greater than the loss of blood, and there is incipient hemoconcentration. The fundamental difference, however, is the great, uncompensated loss of blood. The organism manages, however, in compensation for the loss of blood to draw back plasma-like fluid into the circulation; the blood volume increases again, and the risk of shock disappears. If the organism does not achieve this, the loss of plasma and the hemoconcentration persist and the blood volume falls still lower. A vicious circle has materialized, leading to irreversible shock and fatal outcome (Figure 5), if effective treatment is not instituted

rapidly, primarily blood transfusions or, as a preliminary measure until blood is procurable, plasma or dextran. This patient might perhaps have been saved if, on the day following the operation, he had been given 2 liters of blood instead of plasma and dextran, which were given because the high hemoglobin percentage had proved confusing.

It is a familiar fact that a manifest shock may be relieved by infusion of dextran. This does not mean that dextran is the most suitable agent, however. Blood is lost, and it is this that has to be replaced. If shock is immanent or if the patient has been in manifest state of shock, with loss of plasma from the blood stream, this is sucked back again when the shock is relieved by blood transfusions.

There is even a possibility that dextran has a directly unfortunate effect on the spontaneous defensive reactions set up by the organism when there is loss of blood, so that more plasma is drawn back into the circulation at the expense of the hemoglobin production (Figure 4). I have shown previously ‡—and it is also made evident by these investigations—that strong forces are at work to retain or restore normal blood status, and it is not unlikely that direct intervention with artificial agents (e. g., dextran) may be disturbing. This does not mean that dextran should not be resorted to as a substitute for blood or to increase the blood volume in cases in which blood is not procurable sufficiently quickly or not in an adequate amount. As soon as possible afterward, however, the anemia should be treated.

Obviously, strong forces are being advanced to maintain a normal blood volume, and the hemoglobin and the serum proteins belong to the staff. The serum proteins and the plasma volume constitute the light mobile units which may be advanced rapidly when need arises for increase of blood volume, and they may be withdrawn equally rapidly. The hemoglobin and the red cell volume are more heavily mobile. If these become lost, the organism requires a long time to effect compensation. Once they have entered the blood stream, however, they remain there longer. Together they supplement each other in a fascinating interplay to maintain the blood volume.

‡ References 2 and 3.

SUMMARY

The hemoglobin percentage, the total hemoglobin, the blood volume, the red cell volume, and the plasma volume and also the serum protein concentration and the total circulating serum protein have been examined preoperatively and postoperatively in patients with gastric carcinoma.

In these patients is generally found a considerable reduction of hemoglobin percentage, total hemoglobin, and red cell volume, as well as a moderate reduction of blood volume. The serum protein concentration is distinctly reduced, but, owing to slight or no reduction of plasma volume, the total circulating serum protein is only slightly reduced.

To bring the patients up to normal values preoperatively, considerably more blood has to be supplied than can be calculated from the values found, because a large portion of the red blood cells given are hemolyzed and relatively still greater amounts of serum protein and fluid leave the blood stream again. The result of the apparent excess dosage is that the blood volume remains almost constant (Figures 1 and 7).

Postoperatively, the values are again thrown out of balance, and by investigation of blood values in uncomplicated gastric resections for ulcer in which no blood transfusions had been given, the patients having been put on standard fluid regimen curves have been prepared for the values examined, which may be regarded as "standard curves" in an uncomplicated gastric resection (Figure 2). Twenty-four hours postoperatively the loss of blood found by examinations of total hemoglobin is five times greater than that found by the weighing of compress blood.

Similar relations have been found in subtotal gastrectomy for carcinoma, although

the findings do not warrant such definite conclusions, because of greater variations in the surgical procedures and because blood transfusions have been given before as well as after the operation (Figure 3).

In abdominothoracic total gastrectomy with splenectomy and pancreas resection, considerable deviations are found, the loss of blood during the operation and the following 24 hours being considerably greater than in subtotal abdominal gastrectomy. Otherwise, an essential difference exists from abdominal subtotal gastrectomy, in that the fall in plasma volume postoperatively is relatively greater than the fall in the red cell volume, which is looked upon as an expression of a state of preshock with abnormal permeability of the vessels to plasma-like fluid. As the preshock is overcome, the plasma volume again rises more rapidly than the cell volume. The fall in plasma volume and, consequently, in blood volume, may be diminished by administration of dextran (Figures 4 and 5).

If manifest shock develops, changes take place of a similar kind as in preshock, only still more marked (Figure 6).

In conclusion, the loss of blood during gastrectomy and the following 24 hours is considerably greater than that measured by the weighing of compress blood, usually several times greater. The hemoglobin percentage and the serum protein concentration are poor guides as to the amount of blood lost, as they reach the lowest value two days or several days after the loss of blood. The best guide is provided by measurement of the total hemoglobin and the red cell volume. The serum protein and the plasma volume are more easily mobile; they pass more easily forwards and backwards between blood vessels and tissues, giving a less reliable picture of the loss of blood.

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Gastric Carcinoma Treated with Abdominothoracic Total Gastrectomy

BJARNE FRETHEIM, M.D., Oslo

Removal of the tumor-bearing organ together with the regional lymphatic system is a principle in carcinoma surgery. This was not sufficiently clearly formulated when Conner,[2] in Cincinnati, made the first total gastrectomy, in 1884. His patient died on the operating table, and the first total gastrectomy to succeed was performed by Schlatter [16] in Switzerland in 1897. A classic description of the lymph drainage of the stomach was given by Jamieson and Dobson,[8] in 1907, and subsequently a series of detailed investigations have been carried out in this field—in this country by Eker *—which forms the basis of block dissection in total gastrectomy. Block dissection gradually has become more radical, in that the great and the lesser omentum have been included, as well as the peritoneum in the omental bursa, the spleen, and, frequently, also the pancreatic tail and body. It has also been demonstrated that carcinoma in the upper part of the stomach very easily spreads to the lower part of the esophagus. Resection of the esophagus, therefore, should be performed at an ample distance from the cardia. Downward, on the contrary, the pylorus constitutes a certain barrier against the spread of the tumor.

Two main methods have been used for restoration of the continuity, viz., esophagojejunostomy, with various modifications, and esophagoduodenostomy, the former being the predominant method of anastomosis.

From The University Hospital (Rikshospitalet), Department A of Surgery (Head: Prof. Leif Efskind, M.D.).

* References 3 and 4.

MATERIAL

Our material, which comprises the period 1946 to 1952, inclusive, is made up of 178 cases in which total gastrectomy was carried out through an abdominothoracic incision. The operations have been performed by Johan Holst (since deceased), Leif Efskind, and me. The series comprises 46 women and 132 men, two-thirds in the age group 50 to 70 years. Seven of the patients had been operated on previously for gastric or duodenal ulcer and three patients for polyposis. Average duration of symptoms has been seven and one-half months.

Ninety per cent of the patients were anacid on examination by Ewald test meal. Positive benzidine reaction in feces was found in 83%. In all cases the diagnosis of definite or probable tumor infiltration in the stomach was established by x-ray examination. However, in 12% of the cases, the diagnosis was arrived at only by the second examination, and in 10% of the cases after three or more x-ray examinations.

Two-thirds of the patients had more or less marked anemia. One-third of the cases showed hypoproteinemia, with a tendency toward reduction of the albumin-globulin ratio, and the inverse of this ratio was found in one-third of the cases. The prothrombin percentage showed a tendency to be low.

Tumor infiltration of the entire stomach was found in 29%, infiltration of the cardia and the fundus in 22%, infiltration in the corpus in 43%, and infiltration of the canalis area in 6%.

Table 1 shows the spread of the tumor extragastrically or through the whole of the gastric wall, with subserous nodes on the surface of the stomach. It appears from Table 1 that in 17% of the cases lymph node metastases have not been found, though

TABLE 1.—*Spread of Tumor in 178 Cases of Gastric Carcinoma Treated by Total Gastrectomy*

Localization	Cases, %
Esophagus	22
Duodenum	1
Neighboring organs	24
Resection border	12
Subserous nodes on stomach	22
Metastases to omentum	10
Peritoneal metastases in omental bursa	3
Regional lymph node metastases	83
Tumor adherent to posterior abdominal wall	22

TABLE 2.—*Histological Type of 178 Cases of Gastric Carcinoma Treated by Total Gastrectomy*

Type of Carcinoma	Cases, %
Adenocarcinoma	53.0
Muciparous scirrhus	29.5
Colloid carcinoma	6.0
Undifferentiated carcinoma	5.0
Carcinoma simplex	4.5
Uncertain type	0.5
Squamous cell carcinoma	0.5
Reticulosarcoma	1.0

in three of these cases infiltration was revealed of neighboring organs, viz., in one case of the left hepatic lobe, in another case, of the pancreas, and in a third case, of the resection border on the esophagus.

Table 2 shows that more than half the cases are adenocarcinoma and that somewhat less than one-third are muciparous scirrhus. Gradation according to Broders' index shows that only 10% are of Grade 2; the remainder are of Grade 3 or 4 (Table 3).

OPERATIVE TECHNIQUE

Abdominothoracic incision in right lateral decubitus by the method of Humphreys [7] has been utilized, with splitting of the diaphragm in the direction of the incision as far as the esophageal hiatus. The great and the lesser omentum and the spleen are included as a matter of routine in the block dissection, and one of the operating team (Efskind) has also recently made routine resection of the tail and body of the pancreas. Esophagoduodenostomy has been performed in nine cases, and in the remaining cases esophagojejunostomy has been carried out, as a rule with Braun's enteroanastomosis. In 16 cases one of the team (Efskind) occluded the afferent intestinal loop between the esophagojejunostomy and the enteroanastomosis. Furthermore, resection of the neghboring organ has been carried out to the extent that the tumor infiltration has made it necessary.

TABLE 3.—*Gradation According to Broders of 178 Cases of Gastric Carcinoma Treated by Total Gastrectomy*

Classification	Cases	
	No.	%
Grade II	18	10
Grade III	80	46
Grade IV	76	44
Nongraded	4	..

RATE OF OPERATIVE MORTALITY

Table 4 shows the rate of operative mortality in total gastrectomy in our own material compared with a number of results collected from the literature. Among these, however, only the material of Valdoni [20] consists exclusively of abdominothoracic total gastrectomy, carried out by a procedure similar to that applied in our cases. In our material the main cause of death has been infection originating in the operative field (11 cases). Two patients have succumbed to pneumonia, two to lung emboli, and two to shock, while five patients have died of cardiovascular complications. Two of the remaining patients died of incarceration of the jejunal loop in the slit in the diaphragm, and in neither of these two cases had enteroanastomosis been performed. Two patients died of intraperitoneal hemorrhage, one of them

TABLE 4.—*Rate of Operative Mortality After Total Gastrectomy for Gastric Carcinoma*

Authors	Year of Publication	Operations, No.	Operative Deaths	Rate of Mortality, %
Collected 1884-1942 [13]	1943	294	110	37.4
Pack, McNeer, and Booher [14]	1947	41	13	31.7
Ransom [15]	1947	60	14	23.3
Smith [18]	1947	89	26	29.1
Scott and Longmire [17]	1949	58	6	10
Finsterer [5]	1950	140	80	57.1
Lahey and Marshall [9]	1950	127	31	24.4
Lahey [10]	1950	58	6	10.3
Troell [19]	1950	42	21	50
Valdoni [20]	1951	51	14	28
Walters, Gray, Priesley, and Waugh [21]	1952	62	8	12.9
West and Fenger [22]	1952	60	14	23
Nicolaysen *	1953	45	14	31.1
Own material	1953	178	31	17.4

* Personal communication to the author, 1953.

during heparin treatment for thrombosis. Two patients died of acute pancreas necrosis. Three patients died three to four weeks postoperatively, and autopsy demonstrated extensive metastases.

POSTOPERATIVE NONFATAL COMPLICATIONS

In our series the postoperative complication with the highest incidence has been thromboemboli, which occurred in 10% of the cases. The next highest has been diarrhea, which has set in, as a rule, three to four days postoperatively and with duration ranging from a few days up to two or three weeks. No special explanation has been found for the diarrhea.

In four cases, sutural insufficiency and fistula formation occurred; the fistula closed spontaneously in three cases; in one case it persisted during the six months that the patient survived after discharge from the hospital.

Subphrenic abscess occurred in six cases and pleural empyema in two cases, all of which were cured by drainage. In six cases auricular fibrillation occurred postoperatively. In three cases manifest shock developed. Several of the patients were in a state of preshock postoperatively; however, in all these cases they were treated successfully with blood transfusions. Increased temperature with no definite causation was found in seven cases, and jaundice occurred in four cases. One of these latter patients died four months after discharge, the case being interpreted as one of inoculation hepatitis.

With regard to fatal as well as nonfatal cases, therefore, complications arising in the operative field dominate the picture, and most frequently they are due to sutural insufficiency. In four of the nine cases in which esophagoduodenostomy has been undertaken, sutural insufficiency has occurred. Two of three patients on whom esophagojejunostomy without enteroanastomosis had been performed died of intestinal obstruction. We have been more reserved, therefore, in performance of this operation if it cannot be effected without the least tension, and we also invariably make an enteroanastomosis in esophagojejunostomy.

Relaparotomy has been made for sutural insufficiency in six patients during their initial stay in the hospital. They all died postoperatively, however. Nine patients were readmitted, and relaparotomy was performed, in one case because of troublesome regurgitation to the esophagus. A stenosing operation was done on the afferent intestinal loop, with transitory improvement. The remaining six patients were subjected to relaparotomy because of symptoms of stenosis corresponding to the anastomosis. Five of these cases were caused by recurrence of the carcinoma, while the sixth—in which esophagoduodenostomy had been performed—was due to cicatricial stricture of the anastomosis.

TABLE 5.—*Postoperative Period of Survival for 135 Gastrectomized Patients Discharged from Hospital for More than One Year*

Less Than 1 Yr.	1-2 Yr.	2-3 Yr.	3-4 Yr.	4-5 Yr.	5 Yr. or More
72 of 135 (53.3%)	63 of 135 (46.4%)	25 of 98 (25.5%)	11 of 62 (17.7%)	3 of 63 (8.3%)	1 of 9 (11.1%)

This could be blocked up, and the patient, who was in a miserable state on admission, with severe pellagra, was discharged in a state of well-bearing after having been taught to dilate the esophagus himself.

LATE FATALITIES

Late fatalities indicate postoperative deaths which occurred after discharge of the patient from the hospital.

Table 5 shows that more than half of the patients subjected to total gastrectomy die within one year after the gastrectomy. One-sixth survive three years after the gastrectomy. When the various types of carcinoma are compared, it is found that one-third of the patients with adenocarcinoma have died within one year postoperatively and that more than two years after the operation 27.4% survive. Well over three-fourths of the patients with muciparous scirrhus die within one year after the operation, and more than two years after the operation only 5.5%

survive. Three of the seven patients with colloid carcinoma died within one year after the operation, while the remaining four survive more than two years postoperatively. None of the eight patients with undifferentiated carcinoma survived for more than two years after the operation, and this also applies to the eight patients with carcinoma simplex and also to the one patient with carcinoma of uncertain type.

Relatively, the patient with colloid carcinoma has the best chance, and next comes the one with adenocarcinoma. Eight of the 11 patients who survived more than three years after the operation had adenocarcinomas, and the remaining three had colloid carcinomas.

In correlation to the gradation by the Broders' system, 75% of the 10% with tumors of Grade 2 survive for more than one year, and more than three years after the operation 41% survive. Of the patients with Broders' Grade 3 carcinoma, 46% die within one year after the operation and merely 9% survive for more than three years. Three-fourths of the patients with carcinoma of Broders' Grade 4 die within one year after the operation, and three years after the operation there were no survivals.

Twenty-nine patients (Table 6) had carcinoma infiltration in the esophagus, and three-fourths of these died within one year after the operation. Three of the patients survived more than three years, and one of them survives after more than four years. In this latter patient carcinoma was demonstrated in the resection border on the esophagus, and on follow-up examination four years after the operation—when he was quite symptom-free clinically—esophagoscopy and biopsy disclosed adenocarcinoma in the anastomosis.

The patients who had duodenal infiltration died within 1 year after the operation.

Only 22% of the patients who exhibited tumor infiltration of other adjacent organs survived for more than one year after the operation, and approximately the same relationship was found in patients who had subserous nodes on the stomach or in whom the tumor adhered to the posterior abdominal wall.

In 23 patients (17%), regional lymph node metastases were not revealed. Three of these died within one year after the operation, but in all of them the operation disclosed infiltration of neighboring organs, viz., in one case the left hepatic lobe, in one case the pancreas, and in a third case tumor infiltration of the resection border in the esophagus. All remaining 20 patients survived for more than one year after the operation. Two years after the operation eight patients survived, and three years after the operation five survived. The only patient in our material who survives more than five years after the operation is a patient with adenocarcinoma belonging to this group.

When comparison is made of all factors relating to the spread of the carcinoma, an

TABLE 6.—*Macroscopic and Microscopic Spread of Carcinoma in 135 Patients Gastrectomized More than One Year Previously, Who Died Within One Year, or Survived One, Two, Three, Four, or Five Years Postoperatively*

Spread of Carcinoma	Died Within One Yr., 72 Cases	Survived More Than One Yr., 63 Cases	Survived More Than Two Yr., 25 Cases	Survived More Than Three Yr., 11 Cases	Survived More Than Four Yr., 3 Cases	Survived More Than Five Yr., 1 Case
Infiltration of esophagus	21	8	5	3	1	..
Infiltration of duodenum	2	..	..	..	..	..
Infiltration of neighboring organ	25	7	2	1	..	..
Infiltration of resection border	11	4	1	1	1	..
Subserous nodes on stomach	23	8	2	2	..	..
Peritoneal metastases	2	1	..	..	..	..
Omental metastases	12	1	..	..	..	..
Nonregional lymph node metastases	3	20	8	5	3	1
Regional lymph node metastases	60	43	17	6	..	..
Tumor adherent to posterior abdominal wall	25	6	2	..	..	..

overwhelming difference is found in the postoperative period of survival for patients with and without spread of the tumor extragastrically. Two-thirds of the patients in whom spread outside the stomach has been demonstrated have died within one year after the operation, while all the 20 patients in whom no extragastric spread of the carcinoma has been demonstrated survived more than one year after the operation.

Primarily, the prognosis is decided by the type of carcinoma and the grade of malignancy, as also by the spread of the carcinoma at the time of the operation.

and 60% of those who survive more than two years after the operation state that the complaints are negligible. This shows that especially patients who already have recurrence and metastases in full development have considerable postoperative complaints. All patients—except one who survives more than three years after the operation—represent their complaints as being very moderate. However, the complaints are not actually so insignificant as a rule, but the patients have been adjusted to a new form of life with reduced demands for well-being and working capacity.

TABLE 7.—*Postoperative Complaints in 135 Gastrectomized Patients Operated on More than One Year Previously Who Died Within One Year or Who Survive More than One Year or Two Years Postoperatively*

Type	135 Patients Operated on More Than One Yr. Previously	72 Patients Who Died Within One Yr. Postoperatively	63 Patients Surviving More Than One Yr. Postoperatively	52 Patients Surviving More Than Two Yr. Postoperatively
Swallowing difficulties	32	17	15	3
Anorexia	39	28	11	2
Nausea	30	22	8	2
Vomiting	24	14	10	2
Regurgitation	51	19	32	14
Pyrosis	47	14	33	12
Pain	73	33	40	16
Dumping	6	1	5	1
Diarrhea	22	13	9	3
Obstipation	18	10	8	3
Other complaints	5	2	3	1
No particular complaints	29	1	28	15

POSTOPERATIVE COMPLAINTS AND GENERAL CONDITION

All patients have been followed postoperatively for the period of survival, and the complications the patients have complained about have been compiled in Table 7.

All the patients have had one or other small complaint after the operation. Altogether, however, 21.5% of the patients state that their complaints are negligible. One of these patients died within 10 months after the operation, and relatives report that he was in good form and did not complain of anything until the two last months prior to his death.

Of the 63 patients who survived more than one year after the operation, 44.4% describe their complaints as only slightly troublesome,

Of the patients who have survived more than one year postoperatively, 57% personally describe the result of the operation as satisfactory, and 84% of those who survive more than two years after the operation also consider the result to be satisfactory. Of the patients who have survived more than two years after the operation, 28% state that they have been fully fit for work, at any rate periodically. They have to eat about six meals daily to chew the food well, and to eat slowly. Two-thirds to three-fourths of the patients eat common fare, and half the patients who survive more than two years after the operation have maintained or even increased their weight in comparison with the preoperative weight.

Follow-up examination of the patients more than one year after the operation shows that the longer the interval after the operation the greater the tendency to development of anemia of hyperchromic type. The total circulating hemoglobin is generally reduced and also the blood volume. This dehydration of the patients gives a false representation of the degree of the anemia measured by the hemoglobin percentage. The serum protein concentration shows small tendency to reduction. At the time immediately after the operation there is a tendency to inversion of the albumin-globulin ratio; this tendency, however, becomes less pronounced the longer the interval after the operation. The prothrombin percentage shows a tendency to reduction.

COMMENT

The relatively poor results obtained by subtotal gastrectomy for carcinoma are partly due to the insufficient radicality of the gastric resection, resulting in local recurrence, and partly due to the lymph node dissection not being sufficiently comprehensive or to the lymphogenous metastases having extended outside the area accessible to radical dissection. This appears distinctly from investigations carried out by Coller and collaborators,[3] McNeer and collaborators,[11] and Eker.[4] According to these investigations, it appears as if the cause of the recurrence is as frequently lymphogenous metastases to glands situated outside the field of radical block dissection as it is local recurrence in the stomach. Radicalization of surgery with total gastrectomy and more extensive lymph node dissection is a natural consequence of this observation. The survey shows that a combined abdominothoracic incision is best and that this affords the greatest possibilities for radical block dissection.

The rate of mortality in total gastrectomy, however, was prohibitive for a lengthy period, and not until recent years has it come down to a level justifying discussion in earnest of total gastrectomy as treatment of gastric carcinoma.

During the past 10 years the rate of operative mortality in subtotal gastrectomy carried out in our department through an abdominal incision has been 8.3%, and for the past two years, 6.2%. The rate of mortality here, therefore, is essentially lower than in abdominothoracic total gastrectomy for carcinoma, which is 17.4% in our material. Even though the rate of mortality has been reduced somewhat during the past two years, it is still double that for subtotal gastrectomy.

Respecting the nonfatal severe postoperative complications, the difference in disfavor of the total gastrectomy is still greater. It is true that some complications are directly due to technical errors, viz., esophagoduodenostomy performed with too great tension and, in esophagojejunostomy, failure to place an enteroanastomosis. Even though these cases are disregarded, however, the incidence of complications in total gastrectomy is considerably higher than that in gastric resection. Though the operative technique has now been well standardized and the preoperative and postoperative treatment has been improved, the number of postoperative complications remain materially higher in total abdominothoracic gastrectomy than in gastric resection.

TABLE 8.—*Postoperative Period of Survival after Subtotal and Total Gastrectomy for Carcinoma*

Operative Method	Percentage of Survivals After		
	1 Yr.	3 Yr.	5 Yr.
Subtotal abdominal gastrectomy	62.6	36.1	22
Total abdominothoracic gastrectomy	53.3	17.7	11.1

Table 8 demonstrates that the postoperative survival time for patients in whom abdominothoracic total gastrectomy has been performed is considerably shorter than for those in whom subtotal gastrectomy has been carried out.

The survival time for patients in whom gastric resection or subtotal gastrectomy for carcinoma has been performed, however, cannot straight off be compared with the sur-

vival time for the patients in whom total gastrectomy has been performed, since the latter figure represents patients with advanced cases of carcinoma, in whom most frequently it has not been possible to do anything but total gastrectomy if radical operation was to be attempted at all.

The patients who have died within one year after total gastrectomy have generally had severe complaints during their period of survival mainly in the form of anorexia, nausea, and vomiting. They have not succeeded in increasing their weight, have been unfit for work, and frequently have been troubled by considerable pain, especially after meals. On the whole, it must be stated that so far as this group is concerned, which is represented by more than half the cases in our series, the operation has not been indicated.

Conditions are much better respecting patients who have survived more than one year after the operation, and still better have those fared who have survived more than two years after the operation, half the number of whom have reached or even exceeded the preoperative weight, and three-fourths of whom eat practically all kinds of food. One-fourth of the patients state that they are fully capable of work, and half of them are able to perform some work.

It seems that the cause of the material complaints of the patients who die within one year after the operation is not so much the loss of the stomach per se as the fact that they already have metastases in full development. In cases in which recurrence and metastases require longer time to develop, the patient for a shorter or longer time feels considerably better, and the general condition, state of nutrition, and capacity for work are so good—for a shorter or longer period—that the operation must absolutely be considered justified. Their three main complaints are regurgitations, pyrosis, and pain following meals. These complaints are relatively intractable, but they tend to become less pronounced the longer the survival-time.

The cause of regurgitation and pyrosis, besides the loss of the stomach as reservoir, is the loss of the cardiac function. We have endeavored to remedy this by stenosing the afferent loop to the esophagojejunostomy between this and the enteroanastomosis (Efskind) or by making a very wide enteroanastomosis all the way from the duodenojejunal flexure and almost up to the gastrojejunostomy (Fretheim). Both these procedures seem to have minimized these complaints somewhat.

There is definitely no doubt, however, that the patients in whom it has been possible to leave a small stump of the stomach, either of the cardia or the pyloric part, feel better and are able to lead a more normal life. This is feasible in cardiac carcinomas without reducing the radicality of the operation appreciably, by leaving the pyloric part intact. In carcinomas of the canalis and low carcinomas of the corpus, it may be done by severing the left gastric artery at its point of departure, including all fatty tissue with lymph nodes here, in such a manner that the abdominal part of the esophagus and the cardia on the side of the lesser curvature are dissected quite free. The small part left of the gastric fundus is sufficiently nourished from the side of the greater curvature, where, under these circumstances, the spleen and the proximal short gastric vessels are left intact.

The tendency to macrocytic hyperchromic anemia has been repaired by iron medication and injections of liver extract or vitamin B_{12}. On follow-up examination three years after the operation 60% of the untreated cases of total gastrectomy disclose signs of hyperchromic macrocytic anemia.

Nicolaysen and Ragaard [12] carried out nutrition and equilibrium investigations in 18 of our gastrectomized patients. They found that six of the patients were in definitely negative nitrogen balance. They are of the opinion, however, that this negative balance is due rather to insufficient supply of food than to deficient resorption or increased decomposition. The fat resorption, on the other hand, shows a material failure, two-thirds of

the patients having pathological quantities of fat in the bowels. The majority of the patients demonstrate definite negative calcium and phosphorus balance, and they suggest as causal relation the combination of steatorrhea and vitamin D deficiency. It is extremely interesting in this connection that one of our patients had a subclinical tetany with a calcium level in the blood as low as 7 mg. per 100 cc. They also found that a relatively small percentage of the intake of calories is lost as absorption deficiency (30 to 70 calories).

The reason why the patients do not generally manage to put on weight is that they do not get sufficient nourishment; in order to maintain normal body weight they would have to be given more food than their normal requirement. Food tends to be lacking in variety, and they should therefore have extra supply of vitamins. The fluid supply also has a tendency to become too small.

CONCLUSIONS

Total gastrectomy for gastric carcinoma is recommended in all cases in which the neoplasm has spread to such an extent that it is not possible to resect the stomach at a minimum distance of 5 cm. to both sides from the site of the demonstrated carcinoma and in which the possibility exists of radical surgery. In carcinoma of the type of muciparous scirrhus, the macroscopical delimitation is difficult and the indication for total gastrectomy consequently greater.

Total gastrectomy should not be resorted to as a palliative measure.

A combined abdominothoracic incision permits better access and, thereby, a greater possibility of radical block dissection than an abdominal or thoracic incision.

The rate of operative mortality and the incidence of postoperative complications are so much higher in total gastrectomy than in subtotal resection and the postoperative complaints, digestive disturbances, reduced general condition, and capacity for work are so much greater that subtotal gastrectomy is preferable when it can be carried out according to radical principles.

In cases in which total gastrectomy has been the sole possible form of radical therapy, regulation of the dietary and eating customs of the patient and control of the tendency to anemia may permit the patient to have a postoperative period of survival with such moderate complaints and maintenance of such a state of nutrition, general condition, and capacity for work as to justify this operation.

SUMMARY

Abdominothoracic total gastrectomy has been carried out in 178 cases of gastric carcinoma, with a rate of operative mortality of 17.4%.

An account is given of postoperative complications and causes of death. Within one year of operation 53.3% of the patients died from metastases, and 17.7% survived more than three years after the operation.

The patients who died within one year of the operation had considerable postoperative complaints, the operation not having been indicated here, therefore. The majority of the patients who survive more than one year after the operation are so comfortable that the intervention must be regarded as having been justified.

Subtotal gastrectomy should be preferred when it is feasible according to radical principles.

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Surgical Aspects of Splenic Disease

WARREN H. COLE, M.D.
JAMES D. MAJARAKIS, M.D.
and
LOUIS R. LIMARZI, M.D., Chicago

It is well known that the spleen is not an organ necessary for life, but in spite of its seemingly slight importance there are a surprisingly large number of pathologic conditions resulting from its disease. Many of these diseases (e. g., thrombocytopenic purpura and hereditary spherocytic anemia) respond to splenectomy; accordingly there is little controversy regarding indications for splenectomy in these two diseases. However, in numerous other diseases, such as Banti's disease, acquired hemolytic anemia, and secondary thrombocytopenic purpura, results are inconstant and unpredictable; rarely are we able to explain why results are good or bad in the latter two diseases. For this reason there is much need for more study of splenic diseases, in the hope that we can arrive at a better understanding of indications for splenectomy. In the period 1936 to 1954 we have performed splenectomy on 137 patients with splenic disease. This excludes splenectomy for splenic injury and splenectomy for carcinoma of adjacent organs.

FUNCTIONS OF THE SPLEEN

Removal of a normal spleen results in certain hematologic changes such as a temporary leucocytosis (with mild esophinophilia), thrombocytosis, and, occasionally, erythrocytosis, but rarely is the patient aware of any subjective manifestations. Usually splenectomy produces a decrease in thickness of some of the red cells to such an extent that leptocytic or target cells may be produced. Splenectomy is often followed by a temporary generalized enlargement of the lymph nodes. The removal of the spleen is in no way detrimental to health of the patient.

Normal Functions.—One of the most important of the normal functions of the spleen is the destruction of erythrocytes and conversion of the hemoglobin to bilirubin. This mechanism is brought about by "trapping" of the erythrocytes in the sinusoids of the spleen and conversion of the older cells to spherocytes which are more fragile and thus are destroyed. In fetal life the spleen is active in the formation of all types of blood cells, but in adult life the only remnant of this function is formation of a portion of the lymphocytic and monocytic elements.

The spleen acts as a store house of red blood cells, but this function probably has no significant value to the human being. Much of the iron in the body is stored in the spleen where it is available for the formation of hemoglobin and new red blood cells.

It is thought that the spleen exerts a favorable role in the defensive mechanisms of the body in so far as it may filter certain infectious agents from the blood. There is no definite evidence in the human being that infection is tolerated less well by splenectomized persons. In rats it is well known that after splenectomy the animals' resistance to infection is greatly reduced, particularly to Bartonella infection which will usually ultimately kill the splenectomized rat. There is slight evidence that in animals splenectomy may lessen the resistance to cancer. The observation that shielding the rats' spleen during total body irradiation exerts a protective influence against radiation effects (Jacob-

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From the Departments of Surgery and Medicine, University of Illinois College of Medicine, and the Illinois Research Hospitals.

son [1]) is an important observation on splenic function, but its exact significance is not known.

Abnormal Functions.—In some splenic diseases abnormal functions may result. There are two major theories or mechanisms explaining how these abnormal functions exert their deleterious role. One of these concepts favored by Doan [2] assumes the development of an abnormal degree of phagocytic activity, including the excessive destruction of erythrocytes (hemolytic anemia), platelets (thrombocytopenia purpura) or white cells (primary neutropenia). Another hypothesis favored by Dameshek [3] assumes that the spleen exerts an inhibitory effect on the bone marrow; for example, in thrombocytopenic purpura the inhibitory effect presumably prevents release of thrombocytes from the bone marrow into the blood stream even though bone marrow studies may reveal an excess number of megakaryocytes. This effect may be due to hormonal regulation or to the possible formation of autoantibodies by the spleen and other reticuloendothelial organs. This latter theory is perhaps supported by the demonstration by Harrington and associates [4] that transfusion of blood from patients with thrombocytopenic purpura into normal persons results in a prompt and dramatic decrease in the platelet count of the recipient.

In hereditary spherocytic anemia the fragility of the erythrocyte (particularly the spherocyte) is increased with the destruction of sufficient erythrocytes to produce anemia and jaundice, the latter resulting from the increased formation of bilirubin arising from the hemoglobin of destroyed red cells.

HYPERSPLENISM

By hypersplenism is meant an abnormal and perhaps increased activity of the spleen resulting in a reduction of one or more of the cellular elements of the blood, e. g., thrombocytes, neutrophiles, and erythrocytes. Reduction of just one of these elements may result in the classic diseases such as thrombocytopenic purpura, primary splenic neutropenia, and hemolytic anemia. However, often indeed, more than one element is involved; all three may be reduced, in which case the condition is spoken of as pancytopenia.

The bone marrow is hyperplastic in all types of hypersplenism, and recovery usually takes place after splenectomy. As stated previously, there are two or possibly three explanations for hypersplenism. Doan is of the opinion that overactivity of the phagocytic function of the spleen with increased destruction of one or more of the cellular elements just mentioned is the primary cause of the disease. Damashek and others believe thrombocytopenic purpura and splenic neutropenia are due to an inhibition of the bone marrow by excessive hormonal influence from the spleen. Hereditary spherocytic anemia is thought to be an abnormality of red cell formation, and acquired hemolytic anemia is thought to be produced by formation of auto immune antibodies by the reticuloendothelial system, of which the spleen plays a major role.

Evans and Duane [5] have also expressed the idea that autoantibodies may be found in the spleen and the reticuloendothelial organs and, at times, in the circulating blood, with resultant excessive destruction of some of the cellular elements of the blood. This phenomenon may be observed in acquired hemolytic anemia, thrombocytopenic purpura, and leukopenic states.

Accordingly, there are three current theories or mechanisms for causation of the hematopoietic effects of hypersplenism. However, at the present time we must admit that the explanation of mechanisms still remains conjectural.

Hypersplenism may be primary or secondary. In the former there is no obvious cause whereas in the latter some other disease (e. g., Banti's syndrome, Gaucher's disease, Felty's syndrome, leukemia, Hodgkin's disease, Boeck's sarcoid, malaria, and cirrhosis of the liver) may be the primary factor in production of the hypersplenism. In Doan's series of splenectomies 65% were classified as primary and 35% as secondary.

Years ago most hematologists and surgeons were reluctant to advise or perform

splenectomy in patients with secondary hypersplenism. However, results reported by Zollinger and Williams[6] consisting of 91% good results in primary hypersplenism and 64% good results in patients with secondary hypersplenism indicates that splenectomy has a definite place in the latter condition. However, it is very significant that in their series the postoperative mortality rate in primary hypersplenism was 1.1% compared to 13% in secondary disease.

PATHOLOGIC FEATURES OF SPLENIC DISEASE

In general, gross and microscopic examination of the spleen has been disappointing in clarifying the etiology of the various splenic diseases, and, in fact, has been of surprisingly little help in differentiating one disease from the other. However, a few pathologic features are obvious. For example, the spleen of congestive splenomegaly (Banti's disease) is large, fibrotic, and often adherent to adjacent structures by vascular adhesions. The spleen of hemolytic anemia is large and beefy, but not fibrotic or surrounded by adhesions. In thrombocytopenic purpura the spleen is rarely enlarged.

Microscopic examination of the spleen in hypersplenism reveals phagocytic cells engulfing the formed elements of the blood but there is not agreement that this process is more pronounced than under normal conditions.

Microscopic examination of the bone marrow reveals certain pertinent facts. As already stated, in all of the three conditions under discussion, a hyperplastic bone marrow is present. In thrombocytopenic purpura the megakaryocytes are almost always markedly increased in number, but there appears to be a lack of normal platelet formation, suggesting a maturation arrest. This feature is rather strong evidence against the increased phagocytosis theory and in favor of the inhibitory influence of the spleen on the bone marrow. Moreover, the hyperactive bone marrow of Felty's syndrome with increase in immature granulocytes suggests a maturation arrest or failure of release of the cells from the bone marrow. This finding likewise supports the inhibitory theory.

INDICATIONS FOR SPLENECTOMY

Clinical experience has established the value of splenectomy in the treatment of three main types of hematologic diseases:

1. Hemolytic disorders
2. Hemorrhagic diseases associated with thrombocytopenia in the presence of normal or increased numbers of bone marrow megakaryocytes
3. Hypersplenic states associated with splenomegaly (splenic dysfunction)

On each of these three groups, there is controversy as to whether the peripheral blood changes are due to an influence of the spleen remotely, or the bone marrow (hormonal); to local destruction of circulating blood cells within the spleen (phagocytosis or sequestration); to the presence of a circulating platelet-reducing factor in the blood of some patients with idiopathic thrombocytopenic purpura, or to a combination of several of these mechanisms. In some of the above hematologic conditions even after the bone marrow has returned to normal after splenectomy, the neutropenic and thrombocytopenic elements in the blood may remain depressed. Also the hemolytic mechanism may persist or recur after splenectomy. Thus in some of the above conditions it is apparent that the mechanism responsible for the neutropenia, thrombocytopenia, and acquired hemolytic anemia[7] is not entirely due to a splenic hormone or phagocytic activity of the reticulum of the spleen.

HEMOLYTIC ANEMIA

The disease for which splenectomy is probably more strongly indicated than any other is hemolytic anemia. It may be hereditary or acquired and is usually but not always associated with jaundice, which is produced by the increased destruction of red cells with transformation of this liberated hemoglobin to bilirubin. However, the bilirubin so produced is chemically bound to globin and has certain qualities not making it susceptible to excretion by the kidneys; thus the jaundice is acholuric. The increased production of bilirubin, excreted by the liver into the intestinal tract, is converted by bac-

terial action into urobilinogen. Part of the urobilinogen is absorbed from the intestinal tract into the blood stream from which it is excreted by the liver and kidneys. If liver function is normal, the amount of urobilinogen in the urine roughly parallels that in the stool. If there is impairment of liver function there will be a retention of urobilinogen in the blood stream with increased urinary excretion, even if only a normal amount is absorbed from the intestinal tract.

Hereditary Hemolytic Syndromes.—Although spherocytic anemia is the most common and important disease in this group, several others may be included. The factors common to all are the hereditary tendency and hemolysis.

Spherocytic anemia is transmitted as a Mendelian dominant and occurs in both sexes. The primary abnormality is spherocytosis with increased mechanical and osmotic fragility. Accordingly we need not assume the spleen is diseased, since a normal spleen might destroy enough red cells to produce the anemia and jaundice.

Manifestations are quite variable and may be so mild that they are not recognized. Often there will be a history of occasional attacks of jaundice in childhood and early adult life. In adult life there may be no evidence of disease other than a mild splenomegaly and mild anemia. In doubtful cases inquiry should be made regarding the history of jaundice, splenomegaly or splenectomy in other members of the family. If there is doubt, blood studies should be carried out on them. When the disease is more severe, jaundice is usually constant and the spleen enlarged so much that it is always palpable. Occasionally leg ulcers similar to those observed in sickle cell anemia are encountered. Often the anemia is so marked that weakness and lassitude are prominent complaints. When the disease is still more severe, crisis may develop. In this stage of the disease, as discussed later, chills and fever may be present.

The most characteristic abnormality in the blood is the presence of microspherocytes (red cells which are abnormally thick but having a diameter smaller than normal). As stated, there is an increase in mechanical and osmotic fragility of the red cells. Reticulocytosis (at times up to 15 or 20%) and increased urobilinogenuria are usually present. One-half to two-thirds of the patients have gallstones. The bone marrow reveals a normal hyperplasia, but this finding is not diagnostic.

Occasionally one encounters a chronic hereditary nonsperocytic anemia particularly in children.[8] In these patients there is a hyperactive bone marrow with reticulocytosis and normoblastosis; hyperbilirubinemia is persistent. Mechanical fragility is increased, but saline fragility is normal. We had three such patients in our series.

There are several other types of hereditary anemia for which splenectomy may at times be indicated. Among them are Mediterranean anemia, sickle-cell anemia, ovalocytosis, and congenital porphyria. Splenectomy may be indicated in these diseases when there is an associated hypersplenic state which is rare.

Acquired Hemolytic Anemia.—This type of anemia is caused by extracorpuscular mechanisms (chemicals, drugs, bacteria, and immunologic agents), resulting in destruction of red cells, which usually have a normal osmotic reaction. This prominent difference between the two types of anemia (i. e., hereditary and acquired) was proved by Loutit and Mollison,[9] who found that red cells transfused from patients with hereditary anemia into normal recipients survived only 20 to 50 days, whereas red cells from patients with acquired anemia transfused into normal recipients survived the normal length of time, i. e., 100 to 120 days. Acquired hemolytic anemia may be of two types. In one the cause is unknown; accordingly, it may be designated as idiopathic. The other type may be secondary to some disease or other toxic agent; we have identified this as symptomatic acquired anemia.

In idiopathic acquired hemolytic anemia Evans and associates[10] have consistently found autoantibodies of the agglutinin or hemolysin type. It has been found that even though these abnormal antibodies may not

be demonstrable in the plasma, the washed red cells will be agglutinated by anti-human-globulin rabbit serum; this would indicate that the red cells have absorbed some type of immune antibody on their surfaces. The recent introduction of the Coombs' test (human antiglobulin test) has materially aided in differentiation of hereditary spherocytic anemia and the "autoimmune" type of acquired hemolytic anemia. In the acquired hemolytic anemia, "autoantibodies" can be demonstrated on the red blood cells and in serum by the use of the newly developed techniques.

The manifestations of hemolysis are similar to those encountered in hereditary spherocytic anemia but are more likely to appear later in life and be severer. Complete spontaneous recovery occasionally occurs in this disease but does not occur in hereditary anemia. The spleen is palpable in about half the cases, whereas it is constantly enlarged in hereditary spherocytic anemia. Spherocytosis is present in about half the cases, whereas it is constantly present in hereditary spherocytic anemia. Splenectomy is followed by good results in one-half to two-thirds of the cases, whereas a cure is always obtained in hereditary spherocytic anemia by removal of the spleen. For the acute and subacute forms ot this disease removal of the spleen should be considered when repeated blood transfusions and the administration of pituitary corticotropin (ACTH) or cortisone have failed to produce a remission. In chronic forms associated with recurrent crises, severe uncontrollable anemia, or the formation of gallstones, splenectomy is indicated after corticotropin and cortisone therapy have failed to produce a satisfactory remission.

In symptomatic acquired hemolytic anemia a primary factor is demonstrable. In our series of six patients classified in this group three had a lymphoma and two had leukemia. Other types of malignant disease may likewise give rise occasionally to this type of anemia. Other factors such as chemicals (e. g., lead and sulfonamides), and physical agents such as burns and x-ray have been reported as causative factors but we observed none in our series. Splenectomy may relieve the hemolytic anemia but will have no influence on the primary disease.

Hemolytic Crisis.—This complication may develop in practically all types of hemolytic anemia; it is serious and a definite threat to life. Rapidly increasing jaundice with a falling red cell count should suggest development of a crisis particularly if prostration is present. Chills and fever indicate full development of a crisis which is more serious if oliguria or anuria develops.

Damashek and Taylor [11] believe that crisis is due to a "combination of (1) marked exaggeration of the usual hemolytic mechanisms with (2) arrested maturation of red cells in the bone marrow induced by a pathologically hyperactive spleen." Extreme spherocytosis and increased red cell fragility were present in all of their patients with crisis. The bone marrow is hyperplastic. On the other hand there is evidence that the crisis may result from a temporary hypoplasia of the erythrocytic elements in the bone marrow.[12] This so-called "aplastic" crisis is associated with complete cessation of the formation of the red blood cells; the reticulocytes disappear from the blood. Jaundice decreases and serum bilirubin as well as urobilinuria recede to normal values, whereas serum iron increases. This period is further characterized by leukopenia and thrombocytopenia. Spontaneous recovery (when it occurs) is the result of a rapid regeneration of the erythropoietic tissue. Crisis does not occur in the absence of a spleen.

Opinions differ as to the use of transfusions in the treatment of crisis. Damashek and Taylor [11] favor the use of fresh whole blood. Other observers consider transfusions contraindicated because of the occasional development of a severe hemolytic reaction and resort to immediate splenectomy; blood is started as soon as the splenic artery is ligated. Perhaps a safe compromise is to start administration of blood, give 50 cc. slowly, discontinue for one hour, and then resume the transfusion if no reaction develops.

It is now known that there is a definite beneficial effect from corticotropin and cortisone in acquired hemolytic anemia. One of these drugs should be tried if crisis develops.

THROMBOCYTOPENIC PURPURA

This disease is characterized by excessive bleeding from mucous membranes and various body orifices, as well as into the skin, subcutaneous tissue, and numerous organs of the body. There is a defect in the clotting mechanism or an increase in the permeability of the capillary walls or both; the defect in the clotting mechanism is related to the decreased number of platelets in the blood. The disease is commoner in females and in persons between 10 and 40 years of age. Thrombocytopenic purpura may be (1) idiopathic or (2) secondary to some other factor such as drugs (arsenicals, quinine, gold salts), leukemia, Gaucher's disease, sarcoid disease of the spleen, certain infections (such as tuberculosis), x-ray therapy, extensive burns, and food allergy. Some of the secondary purpuras are not associated with thrombocytopenia.

The mechanisms of the development of thrombopenic purpura are not clearly understood; there are three theories: (1) Wiseman and Doan [13] support the theory that the disease results primarily from excessive destruction of platelets by the spleen; (2) Damashek and associates [3] believe the thrombocytopenia results from inhibitory action of the spleen on the bone marrow (failure of maturation and release of megakaryocytes into platelets); (3) Evans and associates [10] believe the platelets are destroyed by an autoantibody produced by the spleen and perhaps other tissues of the body. The latter theory is supported by the dramatic observations of Harrington and associates,[4] who noted that when citrated whole blood from patients with thrombocytopenic purpura was transfused into normal persons, there was a prompt decrease in the platelet count of the recipient. This effect persisted five to seven days, and in the severe cases there was a prolonged bleeding time and decreased prothrombin consumption.

The laboratory findings are rather characteristic. The blood platelet count may be reduced to 50,000 per cubic millimeter, or lower. The bleeding time is prolonged but the clotting time normal. There is no clot retraction. Bone marrow studies reveal an increase in megakaryocytes.[14]

Classic manifestations are bleeding from body orifices or into the skin (petechia and ecchymosis) or subcutaneous tissues, anemia, weakness, and loss of weight. The spleen is rarely enlarged; if enlarged, some other disease, such as leukemia, should be suspected. Death may be caused by hemorrhage alone, but, as has been emphasized by Learmonth,[15] the danger of hemorrhage into the brain (usually fatal) is so great that this factor alone may be enough to justify splenectomy in all cases, once the diagnosis is established; this judgment is supported by the fact that splenectomy results in cure in such a high percentage of cases.

Splenectomy in idiopathic purpura is followed by good results in about 80% of cases. Up until a few years ago it was thought that splenectomy was contraindicated in secondary purpura. Recently a few cases of cure from splenectomy in secondary purpura have been reported. For example, in 10 patients reported by Elliott and Turner,[16] 6 had complete arrest, 3 sustained doubtful benefit, and 1 died three months after operation. All of these 10 patients had a low platelet count (4,000 to 55,000). The patients reported by Elliott and Turner had one of four diseases or conditions, namely, Boeck's sarcoid, tuberculosis, or disseminated lupus or had had gold therapy.

In our series of 41 patients with thrombocytopenic purpura 35 were of the idiopathic and 6 of the secondary type. Of the six secondary purpuras one was due to a drug, aminopyrine (Pyramidon). Two followed sulfathiazole therapy; one followed rheumatic fever, and the other, brucellosis.

The administration of pituitary corticotropin and cortisone in cases of idiopathic thrombocytopenic purpura may produce a remission. The remission is usually of short duration, but some cases may remain in a

remission for a longer time on maintenance doses of these hormones. The administration of these steroids has proved useful in the preparation of patients for splenectomy. As soon as the rise in the platelet count has been sufficient to prevent excessive bleeding, the spleen is removed. A postsplenectomy thrombocytosis may be observed in some cases treated with hormones prior to the removal of spleen.

Both corticotropin and cortisone may have practical though temporary therapeutic value in idiopathic thrombocytopenic purpura: (1). The hormones may be useful in preparing the patient for splenectomy so that the operation can be performed in the presence of an adequate number of platelets of endogenous origin. (2). If splenectomy is not indicated or if the spleen has already been removed, the hormones may temporarily restore the platelets to a normal level during a bleeding episode.

In some patients with idiopathic thrombocytopenic purpura following steroid therapy there will be little or no rise in the platelet count, but the bleeding manifestations cease. Apparently under such circumstances the steroids are affecting the capillaries; accordingly, failure to obtain a rise in the platelet count after corticotropin cannot be considered a contraindication to splenectomy.

In children spontaneous recovery is the rule; thus splenectomy in children should be reserved for cases of uncontrollable bleeding and chronic, recurrent purpura. It is our experience that intracranial hemorrhage is more commonly a complication in patients of the older age groups. The older the patient, therefore, the less justifiable it is to employ expectant treatment. Following splenectomy, the platelets gradually reach a normal level, and there is no reappearance of the bleeding diathesis. In other cases profound thrombocytopenia may recur after operation without the reappearance of bleeding manifestations. Splenectomy is a valuable and effective therapeutic procedure in the great majority of cases of idiopathic thrombocytopenia, in spite of the fact that hematological abnormality may persist, or recur, in varying degrees in small percentage of cases.

HYPERSPLENIC STATES

In this group we have classified the patients with rather obvious hypersplenism. We cannot feel that this heading discriminates very effectively, because in a certain sense hemolytic anemia and thrombocytopenic purpura are expressions of hypersplenism. However, in this group we have listed the primary neutropenias and pancytopenias. There are several types of hypersplenism of the secondary type (e. g., congestive splenomegaly or Banti's disease, Felty's syndrome, and Gaucher's disease) for which splenectomy is often very beneficial. As discussed later, splenectomy alone is now rarely performed for Banti's disease because the hemorrhage resulting from the dilated esophageal varices is rarely cured by splenectomy.

Splenic Neutropenia and Pancytopenia.—In 1939, Wiseman and Doan [13] described a disease consisting primarily of splenomegaly and leukopenia for which splenectomy is curative. At times fever and pain in the splenic region are noted. Often there is an increased susceptibility to infection. The disease represents a selective form of hypersplenism in which only one of the cellular elements of the blood is involved. The bone marrow is normal or somewhat hyperplastic.

A few years later Doan and associates [17] enlarged on the concept of splenic neutropenia because they had observed patients who also had variable degrees of anemia (hemolytic) and thrombocytopenia. They attributed the cause of these deficiencies to an excessive phagocytosis within the spleen. Bone marrow studies reveal a panmyeloid hyperplasia. Pancytopenia may be primary, without an obvious underlying cause, but usually it is secondary to some disease (e. g., Banti's or Gaucher's disease) associated with a splenomegaly. Splenectomy will relieve the hypersplenic defects mentioned but will not alter the course of the primary disease.

It is of interest that splenic neutropenic and pancytopenic states show only a transient and temporary response to corticotropin and cortisone; however, more frequently splenectomy is followed by restoration of the blood to a normal or near normal level.

Secondary Hypersplenism.—Three different diseases associated with some type of hypersplenism may be classified under this heading; they are Banti's disease, Felty's syndrome, and Gaucher's disease; the former is the commonest.

In Banti's syndrome there is perhaps more confusion about the various aspects of etiology and therapy than in any of the other splenic diseases. The term congestive splenomegaly is applied by many to this disease because of the portal hypertension which is so commonly present. The presence of portal hypertension implies there is obstruction to venous flow; this obstruction may be located in the splenic vein, portal vein, or liver itself. It is extremely difficult to differentiate cirrhosis of the liver from Banti's syndrome, but, in general, patients designated as having Banti's disease have marked splenomegaly with fibrosis of the spleen and a minimal amount of hepatic cirrhosis. In a previous publication we arbitrarily separated patients with obstruction of the splenic vein from other in this syndrome, but since proof of obstruction within the splenic vein is often lacking we choose now to classify all of these patients with venous obstruction in one group: namely, Benti's syndrome.

Important manifestations are splenomegaly, anemia, and leukopenia. The spleen is fibrotic and often surrounded with vascular adhesions; esophageal varices are usually present, and hemorrhage from these vessels is common; jaundice and ascites are late manifestations.

Bone marrow studies reveal a myeloid hyperplasia early in the disease and later a marked erythroid immaturity as well as a maturation arrest of the myeloid tissue and an increase in the number of megakaryocytes. In the series studied by Limarzi and associates [18] the platelet count was low in about a third and icterus present in about one-half of the cases.

During recent years there has been a trend toward doing some type of portacaval or splenorenal anastomosis for Banti's disease instead of doing splenectomy alone. However, as Coller and associates [19] have stated, the mortality rate after this type of operation and the uncertainty of a good long-term result are so significant that we cannot routinely recommend such an operation. Most surgeons favoring a shunt consider hemorrhage as the indication and believe that ascites is a contraindication for the operation. For years Blakemore has contended that if a sulfobromophthalein (Bromsulphalein) test reveals a very severe retention of dye, a shunt operation should not be performed. There is one major disadvantage of doing a splenectomy alone for portal hypertension with hemorrhage, namely, that a splenorenal shunt will not be possible afterwards because the splenic vein is obliterated by thrombosis. The advantages of splenectomy in Banti's disease have been discussed by Durham.[20]

Originally we thought that if splenectomy was performed before cirrhosis developed, the hepatic disease might be prevented; however, we have now observed two or three patients who had splenectomy performed before gross hepatic disease had developed, but years later they developed hepatic disease of the cirrhotic type. Such an observation complicates the picture somewhat and offers evidence that at least in some cases the disease is in the liver primarily, though the gross appearance of the liver and liver function tests may be normal early in the disease.

When bleeding from an esophageal varix develops, splenectomy will be effective only in patients in whom the coronary vein empties into the portal or splenic vein proximal to the point of venous obstruction. From experience gained during the past few years we have learned that the best treatment for

an acute hemorrhage from an esophageal hemorrhage is not operative but is compression with a Patton tube. After the bleeding has been controlled and oral intake of food resumed, some type of a shunt should be considered. If bleeding cannot be controlled by a compression tube, resection of the cardia should be considered. Patients with acute hemorrhage will tolerate this operation but not a shunt with any degree of safety.

In Felty's syndrome the important manifestations are chronic rheumatoid arthritis, anemia, leukopenia, and splenomegaly; it may readily be classified as a secondary pancytopenia. Although not all patients with this disease have thrombocytopenia, two of our five patients had it. Lymphadenopathy and a yellowish-brown pigmentation of the skin are occasionally noted. The bone marrow is hypercellular, primarily involving the granulocytic elements. The results of splenectomy as recorded in the literature are considered variable, but our results have been quite gratifying, although the bone and joint pathology is unchanged.

Gaucher's disease is a rare disease encountered usually in young girls. The important pathologic feature of this disease is deposition of a large amount of lipoid material in the spleen, bone marrow, and lymph nodes. Reticular cells and foam cells in the bone marrow are fairly diagnostic. Important manifestation are splenomegaly, anemia, and pigmentation of the skin. Late in the disease the liver enlarges. Occasionally pancytopenia develops; if this occurs, splenectomy is indicated, otherwise not unless the spleen is so large as to be uncomfortable or jeopardize life by rupture with little or no trauma.

HYPOPLASTIC ANEMIA

Ordinarily this disease is a contraindication for splenectomy. However, on certain occasions, removal of the spleen apparently results in improvement of the primary condition. Edwards [21] reports splenectomy in four patients, one of whom is alive and well 7 years after splenectomy, the second completely well 10 years after splenectomy, and the third much improved 20 months after operation; the fourth died postoperatively. Coller and associates [19] also call attention to the fact that occasionally patients with hypoplastic anemia are much improved after splenectomy and require less frequent blood transfusions.

MISCELLANEOUS CONDITIONS

Splenectomy may be indicated for cysts and tumors of the spleen, which are rare. About 80% of cysts are false cysts of the hemorrhagic type caused by trauma, inflammation, or parasites, whereas about 20% are true cysts of the dermoid or lymphangiomatous type. Most cysts produce pain in the left upper quadrant and left shoulder. Pain alone may be sufficient to justify splenectomy. Occasionally, hypersplenism is encountered with the cyst; if so, splenectomy may be indicated for this reason alone.

Occasionally infarcts of the spleen and aneurysms of the splenic artery are encountered. The former may be undiagnosed before celiotomy; in one case observed by us emergency operation was performed for severe upper abdominal pain and shock, but nothing was found except the splenic infarct. Aneurysms usually produce marked splenomegaly, but the cause may not be determined preoperatively unless the aneurysm is large.

Occasionally, patients with sarcoidosis and myeloid metaplasia of the spleen associated with myeloscerosis of the bone marrow are improved by splenectomy but usually only if hypersplenism is produced by the primary disease.

During the past few years good results have been reported following splenectomy for porphyria. Important manifestation are splenomegaly, hemolytic anemia, photosensitivity, and various types of porphyrins in the urine. In a patient observed by us, uroporphyrin and coproporphyrin were found in the urine.

There are numerous diseases associated with splenomegaly which are not benefited by splenectomy. However, if hypersplenism develops with a significant degree of panhematopenia, splenectomy may be indicated

to relieve this condition, which may be giving rise to such symptoms as weakness, anorexia, and lassitude. If a severe thrombocytopenia develops and hemorrhage ensues, indications for splenectomy may be urgent.

CONTRAINDICATIONS FOR SPLENECTOMY

Pernicious anemia, polycythemia, Hodgkins disease, and agnogenic myeloid metaplasia are diseases for which splenectomy is contraindicated. Leukemia is also a disease for which splenectomy is contraindicated, but Edwards [21] reports a patient who lived four years after splenectomy for severe pain in the left upper quadrant secondary to an enormously enlarged spleen. Splenectomy is obviously contraindicated in acute splenic tumors.

In subacute bacterial endocarditis splenomegaly is usually present but splenectomy is contraindicated except in the rare instances in which a massive infarct due to an embolus develops; we had one such case. Mediterranean anemia and sickle cell anemia are also conditions for which splenectomy should not be performed unless a hemolytic factor is prominent. Likewise, splenomegaly due to such parasitoses as trichinosis, filariasis, kala-azar, and distomiasis contraindicate splenectomy except in chronic cases in which hypersplenism is significant.

RESULTS

Hemolytic Anemia.—Of 54 patients in our series with hemolytic anemia 40 had anemia of the hereditary hemolytic type (see Table 1). Of this group 33 were of the spherocytic type; 3 had anemia of the familial nonspherocytic type, and 1 each had Mediterranean anemia, sickle cell anemia, ovalocytosis, and congenital porphyria. Results were good in all of these patients except the one with sickle cell anemia who sustained very little benefit. One of the patients with hereditary spherocytic anemia died nine years after splenectomy but not of her disease. As noted by others, none of the patients with spherocytic anemia were cured of their spherocytosis although all other manifestations were relieved. Splenectomy was performed in one patient with porphyria (girl of 8 years with porphyria since the age of four months) but too recently to determine the ultimate result.

Splenectomy was followed by diminished porphyrin excretion and disappearance or great improvement of photosensitivity. This is believed to be related to diminished erythropoesis in response to reduced blood destruction (Schmid and associates [22]).

Of the 14 patients with acquired hemolytic anemia we have designated 8 as idiopathic with or without autoantibodies. One of these died 15 days after operation of a hemolytic crisis. Three of the eight had a good result after splenectomy. Four had only fair results because of recurrent hemorrhage (in two cases) or recurrent jaundice (in three cases). We have designated six of the patients with acquired hemolytic anemia as symptomatic. One of these had myeloid metaplasia of the spleen; she died of pericarditis three months after splenectomy. Three had a lymphoma of the spleen: two of these had Hodgkins disease and are already dead (one two years after operation and the other three years after operation); the third patient had a lymphoblastoma and is alive one year after splenectomy. Of two patients having splenectomy for leukemia, one died 1½ years after splenectomy whereas the second is still alive but it is only six months after operation.

Thrombocytopenia Purpura.—Of 41 patients in this group 35 were classified as having idiopathic purpura. Results in the idiopathic group were good in 31, or 88.5%, and fair in 3. There was one operative death. This patient had been bleeding from the gums, vagina, and urinary tract for four or five days before operation. After splenectomy she continued to bleed and died 20 hours after operation. No platelets were found in the blood stream before or after operation.

Six of the 41 patients in this group had a secondary thrombocytopenic purpura. Three, or 50%, sustained good clinical results; the platelet count returned to normal in all of these. One had a good clinical result but was classified as having only a fair result because the platelet count rose

TABLE 1.—*Types of Disease for which Splenectomy was Performed* *

Disease	Cases, No.	Results			Operative Deaths	Comment
		Good	Fair	Poor		
I. Hemolytic anemia	54					
A. Hereditary	40					
1. Spherocytic	33	32	1	..	..	
2. Nonspherocytic	3	3	..	..	..	
3. Mediterranean	1	1	..	..	..	
4. Sickle cell	1	..	1	..	..	
5. Ovalocytosis	1	1	..	..	..	
6. Congenital porphyria	1	1	..	..	..	
B. Acquired	14					
1. Idiopathic with or without hemolysins or agglutinins	8	3	2	2	1	One postop. death, hemolytic crisis
2. Symptomatic						
A. Myeloid metaplasia of spleen	1	..	..	1	..	Died of pericarditis 3 mo. after operation
B. Lymphoma	3	1	..	2	..	2 already dead
C. Leukemia	2	1	..	1	..	1 dead 1½ yr. after operation; other alive at 6 mo.
II. Thrombocytopenic purpura	41					
A. Idiopathic	35	31	3	..	1	One patient died 20 hr. after operation; she was bleeding for 4 or 5 days before splenectomy, which failed to stop the hemorrhage
B. Secondary	6	3	1	1	1	
III. Hypersplenic states	35					
A. Primary splenic neutropenia and pancytopenia	4	4	..	..	..	
B. Secondary						
1. Banti's syndrome (congestive splenomegaly)	22	4	3	10	5	8 of the 17 patients surviving operation have died—7 of hemorrhage from esophageal varices
2. Felty's syndrome	8	4	2	1	1	
3. Gaucher's disease	1	1	..	..	..	Patient is alive and well 6 yr. after splenectomy
IV. Hypoplastic anemia associated with cellular bone marrow	2				1	One patient died of pericarditis 3 mo. after splenectomy; the other died 11 days after operation of cerebral hemorrhage with no clinical response to splenectomy
V. Miscellaneous	5					
A. Congenital splenic cyst	2	2	..	..	..	
B. Aneurysm of splenic artery	1	..	1	..	..	Developed cirrhosis (see text)
C. Splenic infarcts (endocarditis)	1	1	..	..	..	
D. Splenomegaly and anemia of unknown cause	1	1	..	..	..	

* Illinois Research Hospital 1936-1954.

no higher than 70,000. Another patient was classified as sustaining a poor result. She had a thrombocytopenic purpura secondary to a drug, aminopyrine (Pyramidon) and was bleeding from the gums and vagina at the time of splenectomy which failed to stop the bleeding. For a time the bleeding was controlled by corticotropin, but after a few courses of the drug it failed to have any beneficial effect. She died from a cerebral hemorrhage four months after splenectomy. The sixth patient who had a preoperative platelet count of 0 to 46,000 and had been bleeding for two days before operation died five hours after operation from a severe pulmonary hemorrhage.

Of the 35 patients having splenectomy for idiopathic thrombocytopenic purpura, 27 have had platelet count determinations performed from 1 to 17 years after splenectomy. Five, or 18%, had a count between 50,000 and 100,000. Twelve, or 44%, had a normal count (100,000 to 250,000), whereas 5, or 18%, had a count above normal. The highest of this group was 800,000, noted five years after splenectomy.

Hypersplenic States.—In this group there were 35 patients; 4 of these who had a primary neutropenia or pancytopenia had a good result after splenectomy.

We have classified 22 patients with Banti's syndrome or congestive splenomegaly in this group. Because results following splenectomy alone have been so poor in congestive splenomegaly we have performed splenectomy on only four of these patients since 1948 and in three of these have performed a splenorenal or portacavel shunt simultaneously. There were 5 operative deaths in these 22 patients, constituting a mortality rate of 22.7%; this is a higher rate than in any other group, and corresponds to the experience of others. Four of the five operative deaths were due to postoperative hemorrhage at the operative site. One died of pneumonia and hepatic insufficiency. Eight of the Banti's group have died since discharge from the hospital; seven of these died of hemorrhage from esophageal varices and one of pneumonia. Of the nine patients still living, seven have had one or more hemorrhages from esophageal varices.

We have performed splenectomy on eight patients with Felty's syndrome. Four of these sustained marked subjective improvement and have normal hematologic findings. Two have sustained only slight improvement in their arthritic symptoms. One patient died one year after splenectomy of a malignant thymoma. The eighth patient developed a platelet count of 5,000,000 a few days after operation and was given heparin and bishydroxycoumarin (Dicoumarol) to prevent thrombosis and pulmonary embolus; on the ninth postoperative day she suddenly developed coma and died. Autopsy revealed a ruptured aneurysm of the right vertebral artery.

We have performed a splenectomy on only one patient with Gaucher's disease. She is alive and well six years after splenectomy; hematologic findings are normal.

Hypoplastic Anemia.—We performed splenectomy in only two patients having this type of anemia. Results were poor in both. One died of pericarditis three months after operation. The other patient died 11 days postoperatively of cerebral hemorrhage. There was no clinical or hematological response following splenectomy. The hematopoietic centers may have been exhausted owing to the long duration (three years) of this disease. However, as has been noted previously in this paper, other authors have reported good results in an occasional patient with this disease.

Miscellaneous Diseases.—Splenectomy was performed on five patients with miscellaneous diseases. Two of these had benign cysts, one an aneurysm of the splenic artery, one a splenic infarct (probably resulting from an endocarditis), and a fifth had splenomegaly with anemia of unknown cause. As would be expected, the two patients with splenic cysts had an excellent result.

The patient with an aneurysm of the splenic artery had a history of bleeding from esophageal varices before splenectomy in

1945. She was well for six years, then had another hemorrhage, indicating recurrence or persistence of portal hypertension. Operation revealed a severe cirrhosis of the Laennec type. Portacaval anastomosis was impossible because of numerous varices surrounding the portal vein which appeared to be absent (probably thrombosed previously). A resection of the cardia was performed; when last seen 2½ years after operation she was well and had had no additional hemorrhages. In a study of 198 cases of splenic artery aneurysm encountered in the literature (including 6 of their own), Owens and Coffey [23] found evidence of portal hypertension in 20%. This incidence is too high to be a coincidence and suggests a relationship (unexplained) between the aneurysm and portal hypertension.

The patient having splenectomy for infarct of the spleen suddenly developed signs of shock with sufficient abdominal signs to justify celiotomy; examination of the spleen after its removal revealed a large clot in the splenic artery. She is alive but still has evidence of endocarditis.

SUMMARY

Although it has been known for years that splenectomy yields very good results in familial hemolytic anemia and thrombocytopenic purpura, it has not been until the past few years that we have learned good results are often obtained in acquired hemolytic anemia and secondary purpura. The indications for operation in these latter two diseases are not well defined, although the presence of some phase of hypersplenism represents the clearest indication. Accordingly, much more experience is needed in these diseases having borderline indications.

In our series of 137 patients having splenectomy for splenic disease, 54 had hemolytic anemia. Of this group, 40 were classified as hereditary and 14 as acquired. The important differentiating feature in these two groups is the fact that in the former there is an increased mechanical and osmotic fragility of the red cells, whereas in the latter the red cell fragility is normal. Conforming to the reports of others, splenectomy yielded good results in all except one of our patients with hereditary anemia. In the acquired group (in which Evans and associates have found autoantibodies, agglutinins, or hemolysins) we performed splenectomy on eight patients but sustained good results in only three.

We performed splenectomy in 41 patients with thrombocytopenic purpura. Good results were obtained in 88% of 35 classified as idiopathic; in 6 cases classified as secondary purpura, results were good in only 3.

Under the heading "Hypersplenic States" we have classified primary splenic neutropenia and pancytopenia, Banti's syndrome, and Felty's syndrome. Conforming to results originally reported by Doan and associates we had good results in all of the first group. For the past several years we have not performed splenectomy (one exception) in Banti's syndrome unless a portacaval or splenorenal shunt was performed at the same time. Results of the shunt operations are not included in this series. We have been able to follow all of our 22 patients having splenectomy for Banti's syndrome. Results were good in only four (18%); the mortality rate was 22% in the Banti group. Eight of the 17 patients surviving operation have died since leaving the hospital, 7 of hemorrhage from esophageal varices. Good results were obtained in four of eight patients with Felty's syndrome. We have performed splenectomy for Gaucher's disease in only one patient; she is alive and well six years after splenectomy.

TABLE 2.—*Mortality Rate in Splenectomy* *

Disease	Cases, No.	Operative Deaths
I. Hemolytic anemia	54	
A. Hereditary	40	0
B. Acquired	14	1
II. Thrombocytopenic purpura	41	
A. Idiopathic	35	1
B. Secondary	6	1
III. Hypersplenic states	35	
A. Primary	4	0
B. Secondary	31	6
IV. Hypoplastic anemia	2	1
V. Miscellaneous	5	0
Total	137	10
Operative Mortality Rate		7.3%

* Illinois Research Hospital 1936-1954.

The mortality rate in the entire series was 7.3%. It is very significant that the mortality rate was lowest in the groups (hereditary hemolytic anemia and idiopathic thrombocytopenic purpura) having the best symptomatic results after splenectomy.

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Adrenal Function in the Combat Casualty

CAPT. JOHN M. HOWARD (MC)
LIEUT. JOHN M. OLNEY (MC)
LIEUT. JOHN P. FRAWLEY (MSC)
CAPT. RALPH E. PETERSON (MC), U. S. Army
and
SERFIN GUERRA, B.S., Washington, D. C.

This study was part of a broad survey of the systemic response to injury, carried out at a forward surgical hospital on the Eastern Front in Korea during 1952. This was a time when the front lines were stable and the flow of casualties was usually limited. Evacuation time averaged three and one-half hours. Because the casualty load was seldom very heavy, patients could be held at the forward hospital until they were ready for evacuation. Intensive studies could be made during this period of relative quiescence.

MATERIALS AND METHODS

Twenty battle casualties were chosen for study during a period of 7 to 14 days immediately after injury. The study was designed to study adrenal function during this period of early convalescence. This was often a period of repeated traumas—initial injury, evacuation, anesthesia, operation, secondary dressings, and secondary débridement. All of the patients studied were critically injured. Several died of their injuries or were evacuated because of post-traumatic renal insufficiency.

The study consisted of daily eosinophile counts [10] at approximately 0800 hours, determination of daily urinary excretion of 17-ketosteroids * and corticosteroids † (formaldehydogenic), and balance studies of sodium and potassium.[5] During the first week of convalescence, most of the patients were on intravenous fluids or on standard canned liquids. Intakes could be rather accurately determined during this period by measurement of intake and analysis of representative samples. During the latter part of a patient's study, representative servings of food were weighed, and electrolyte content was calculated from standard charts (Hospital Diets, Department of the Army Technical Manual 8-500). Thus intake measurements during the latter phase were, of necessity, less accurate than those of the earlier days. Urine, gastric drainage, and other fluids excreted in significant quantities were measured and analyzed. In charting the balance studies, values for the day of injury and operation, although studied, were usually not graphed, because the blood loss measurements were inaccurate. On the day of admission, whole blood and glucose were used almost exclusively.

This report is designed to describe the magnitude and duration of the adrenal cortex response and the changes in electrolyte pattern in the severely injured casualty and to contrast the response in those casualties whose course was uncomplicated by severe infection or wound necrosis with those who developed tissue necrosis and infection. The effects of secondary injury are also demonstrated by many of the studies.

RESULTS

Analysis of the studies indicates the response to repeated trauma. In response to the original trauma, or, more correctly, in response to the continuous trauma of the day of injury, the corticosteroid (formaldehydogenic compounds) excretion rose. This increased excretion continued from one to three days and then subsided. The low rate of excretion was maintained for one to two days and was then sometimes followed by a secondary rise of lesser magnitude. Secondary trauma after the first three days was usually followed by an increased excretion of this fraction. Occasionally there was a late rise of considerable magnitude.

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From the Surgical Research Team in Korea; the Department of Biochemistry of the Army Medical Service Graduate School, United States Army, and Walter Reed Army Medical Center, Washington D. C.

Dr. Howard's present address is Department of Surgery, Baylor University College of Medicine.

*References 3, 4, 5, 11, and 12.

† References 2, 5, and 7.

The validity of this rise might be questioned, but, in view of the repetition of the analysis and the consistency of the test in demonstrating an early rise, this occasional late "spontaneous" rise would appear valid. The only exception to the increase in excretion was in a patient who was developing posttraumatic oliguria. Presumably, this failure in excretion of the steroids was renal in nature rather than adrenal.

The 17-ketosteroid excretion was increased only in an occasional patient.

Without exception, the eosinophile count fell with the original trauma. Thereafter the eosinophile count tended to be inversely proportional to the excretion of the corticosteroids. About the third to the fifth day the concentration rose to a higher level, normal or above, and then fell again. Secondary trauma usually caused a modest secondary drop for a few hours.

The balance studies demonstrated the selective conservation of sodium and the excretion of potassium. The point of interest in these studies is the magnitude and duration of these changes. The massive and repeated traumas sometimes maintained these phenomena throughout a period of 7 to 14 days. The most striking changes, however, were noted in casualties with wound necrosis and infection. Adrenal stimulation was prolonged in the latter group.[10]

The following cases are representative of the 20 patients studied.

CASUALTIES WITH UNCOMPLICATED CONVALESCENCE

PATIENT 1.—This soldier, age 23 years, weight approximately 165 lb. blood Type A, had been in the front lines for two weeks prior to injury. While on patrol at 2:30 a.m., Feb. 18, 1952, he was wounded by a land mine. The injuries included a traumatic amputation of both legs at the level of the calf. Tourniquets were applied to the legs and the patient was given 1000 cc. of blood, 200 cc. of albumin, and 500 cc. of plasma at the Battalion Aide Station.

He arrived via helicopter at the hospital four hours after injury. His blood pressure at this time was 120/80, pulse 96 and of good quality. He was pale, but his peripheral circulation seemed fairly good. After another 1000 cc. of blood, his blood pressure rose to 140/80, pulse rate remaining 96 per minute. When his litter was moved to the operating room, six hours after injury, his blood pressure fell to 110/60. Reamputation of the legs was performed under spinal anesthesia and required one and one-half hours. His course was smooth throughout the procedure except for a mild hypertension. Immediately after operation he was given his third liter of blood. Amputation was associated with a blood loss measured at 1350 cc. There was no detectable blood loss thereafter.

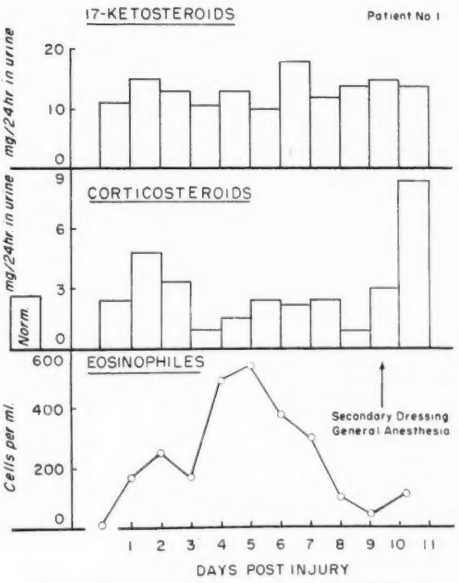


Fig. 1.—The normal excretion of 17-ketosteroids, the increase in excretion of corticosteroids after the primary and secondary operative traumas, and the fall and rebound phenomena in circulating eosinophile concentration. (Normal range of 17-ketosteroid excretion, 8 to 22 mg. per day.)

On the ninth postoperative day, a secondary dressing was performed with the patient under thiopental (Pentothal), nitrous oxide anesthesia. The wounds appeared clean.

The blood given on the day of admission was Type O, 21 to 22 days of age. During the subsequent course he was given a total of 2500 cc. of fresh, type-specific blood. By the fifth day after injury he was eating a liberal diet.

Comment.—This patient's course represents the response to uncomplicated trauma. The pattern of the corticosteroid excretion was one of an initial rise, with a secondary rise on the 10th day after secondary trauma (Fig. 1). The late rise, following a secondary insult, was of greater proportion than

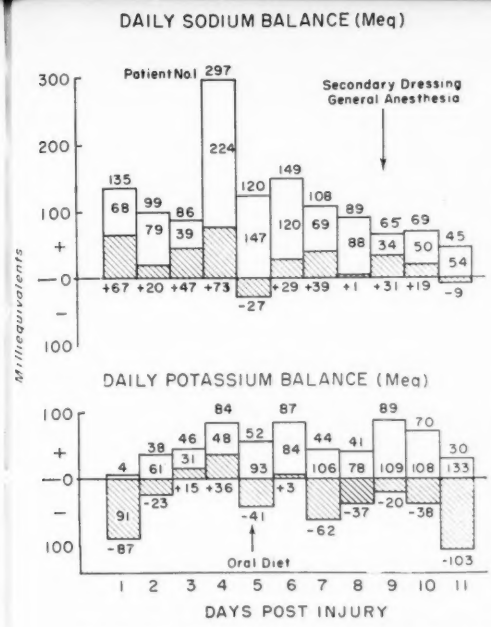
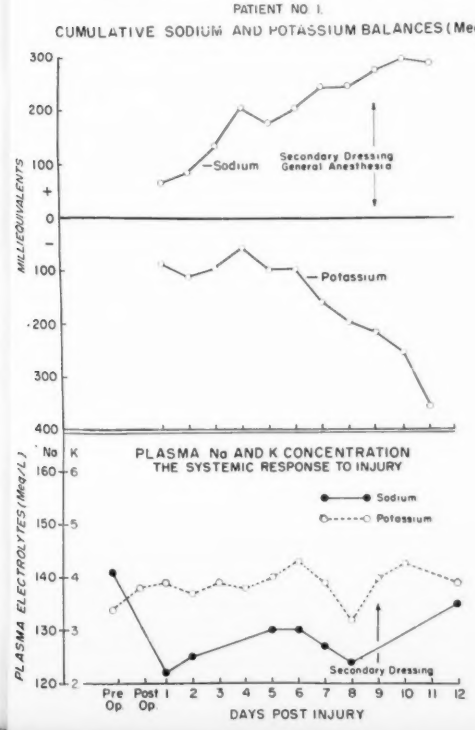


Fig. 2.—These charts are constructed so that the height of the column above the zero line represents intake (indicated by the figure above the column). The figure in the column represents output, and the shaded area and the figure below the column represents the positive or negative balance (after Moore). On the 11th day after the original injury, the patient was still losing potassium and had barely begun to excrete the retained sodium.

Fig. 3.—Persistent retention of sodium and loss of potassium throughout the 12-day period.



the original response. The height of the peak and its duration may be related not only to adrenal stimulation, but to the ability of the kidneys to excrete the steroids. This hypothesis is based on the studies which demonstrated a marked decrease in glomerular filtration after massive injuries.[6]

The 17-ketosteroid excretion remained normal throughout his course. The eosinophile count fell to zero after trauma. As his corticosteroid excretion subsequently diminished, the eosinophile count rose to a peak of 540 per ml. on the fifth day after wounding. This would appear to be a rather definite rebound phenomena,[10] in which the count rose above normal. It rapidly fell thereafter and remained at a low normal level after the secondary operation. His oral temperature remained 98 to 100 F throughout his course except for a spike to 103 on the seventh day.

He remained in positive sodium balance (Fig. 2) throughout the first 10 days of the study except for 1 day. On the fifth day he was in a slightly negative sodium balance. This was the day on which his eosinophile count reached its peak and was one of the days on which the corticosteroid excretion was very low. On the eighth day his balance was almost zero, but he swung back to sodium conservation for two days after the secondary anesthesia. During the first 11 days after injury, therefore, his cumulative sodium retention was approximately 290 mEq. (Fig. 3).

He developed a negative potassium balance for two days which, again, became more pronounced after the secondary trauma. On the 11th day, the day of the maximal corticosteroid excretion, his potassium loss was also maximal (Fig. 2). Thus his cumulative potassium loss was approximately 355 mEq. during the first 11 days (Fig. 3). The plasma sodium concentration fell rapidly over a few hours from 141 to 122 mEq/L. and during the course of the study never reached a normal concentration again, in spite of the sodium conservation. In contradistinction, the potassium diuresis was

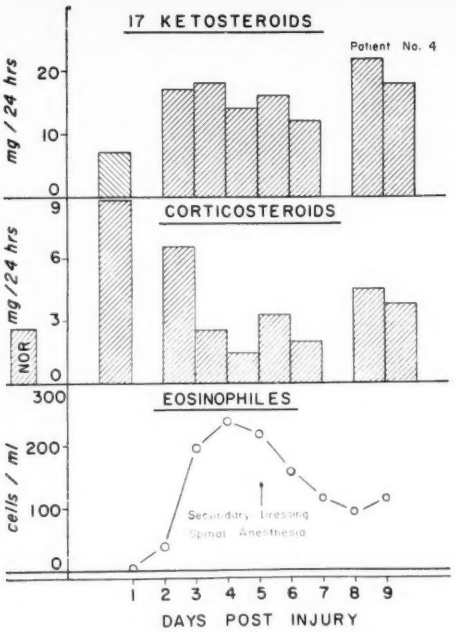


Fig. 4.—The corticosteroid excretion was quite high on the first day and gradually fell to normal. Secondary anesthetic trauma was associated with only a slight corticoid response.

associated with a slight rise in the plasma concentration of potassium after the secondary injury.

Water conservation was noted early and was followed by a diuresis which was only transiently checked by the secondary anesthetic trauma. Thereafter the diuresis continued in spite of the adrenal hyperactivity.

Patient 4.—A 21-year-old American soldier, weight 155 lb. blood Type A, was in Korea two months, and in the combat area the entire time.

This soldier was wounded at 1 a. m., Aug. 19, 1952, by the explosion of an artillery shell. The injury consisted of shattering fractures of both femurs as well as fractures of the pelvis and extensive soft tissue injuries.

At the Battalion Aide Station, 3:15 a. m., his blood pressure was 100/60. He was given albumin 100 cc., blood 1000 cc., isotonic sodium chloride solution 1000 cc., morphine 30 mg. followed three hours later by 8 mg.

On arrival at the hospital via helicopter at 6:30 a. m., his blood pressure was 82/40 and his condition appeared precarious.

By 9:20 a. m. he had received 2500 cc. blood at the hospital. His pressure had risen to 165/96. With the onset of thiopental (Pentothal), nitrous oxide, and ether anesthesia, his blood pressure fell to 105/60, with a pulse rate of 102 per minute.

Toward the end of operation the pressure fell to 82/42, pulse 124 per minute. Operation consisted of débridement and casting of the legs and required one and one-quarter hours. By the end of operation he had been given a total of 12 pt. of blood. His pressure again fell to 80/50, pulse 160. He was then given an additional 6 pt. of blood, 3 units of albumin, and 1000 cc of 5% dextrose in water. His pressure gradually rose to 112/55, pulse 148, respirations 20 per minute (7 p. m.). Because the pressure again fell to 90/50 later during the night, another pint of blood and unit of albumin were given (total 19 pt. of Type O bank blood, 15 days of age, and 5 units of albumin).

His subsequent course was smooth. The blood pressure stabilized by the first postoperative day at 120/60, pulse 140.

A secondary dressing was performed with the patient under spinal anesthesia on the fifth postoperative day. The wounds appeared clean.

Comment.—This patient had a massive primary injury. His subsequent course was smooth. His corticosteroid excretion was elevated early and fell to normal limits on the third and fourth days after injury (Fig. 4). This change was accompanied by inverse changes in eosinophile concentration (Fig. 4) and sodium and potassium balances (Fig. 5). The secondary trauma on the fifth day after injury was followed by only a slight response. The corticosteroid excretion was elevated again at the end of the nine-day study. This repeated adrenal response resulted in a continued conservation of sodium, reaching a level of 876 mEq. at the end of seven days (Fig. 5).

In this casualty with a massive wound without necrosis, the sodium retention was much more marked than was the potassium loss.

Patient 5.—A 19-year-old American soldier, blood Type O, was in Korea two months, in combat two months.

This soldier was wounded at 10:30 a. m., Sept. 1, 1952, by the explosion of a land mine. His injuries included traumatic amputation of both legs at the knee. He arrived at the Battalion Aide Station 30 minutes later and was given 1500 cc. blood and 30 mg. morphine.

On arrival at the hospital (via helicopter) at noon, his blood pressure was almost unobtainable. His radial pulse was faint. After 2500 cc. blood was given intravenously, his blood pressure was distinct at 60/50. Arterial transfusion was then

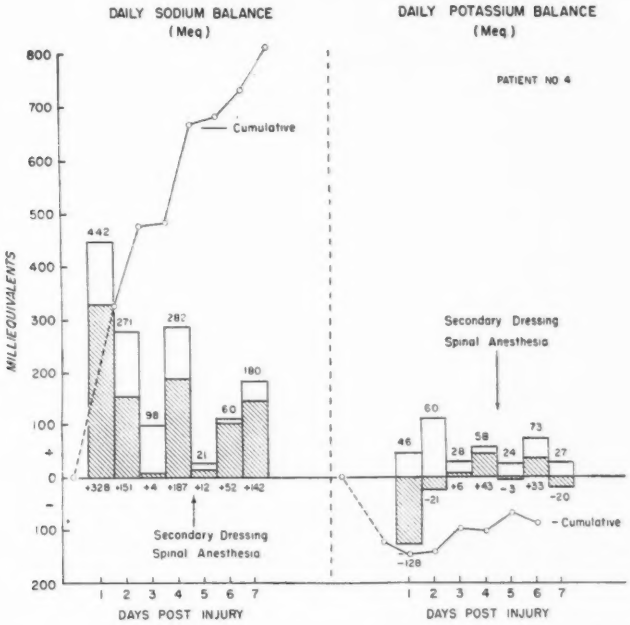


Fig. 5.—The retention of sodium was much more pronounced than the loss of potassium.

begun, and his blood pressure rose rapidly to 115/80 after the infusion of 3500 cc. by this route. After 5000 cc. of blood was received at the hospital, the pressure was 142/100, pulse 136. Thiopental anesthesia was then begun (2:40 p. m.), and the pressure fell to 98/50, pulse rate 140 per minute. With bilateral tourniquets in place, bilateral amputation was performed. The amputations were radical, high in the thigh, proximal to all visibly injured tissue. Of all patients studied, the débridement of this patient was perhaps the most radical and therefore the most complete. Nitrous oxide, oxygen, and ether were used in addition to a total of 175 mg. thiopental.

Operation was completed at 4 p. m. His blood pressure never rose over 100 mm. systolic during the procedure, and the pulse rate ranged about 130 to 140 per minute. By the end of the operation he had received 18 pt. of blood (21 including that at the Aide Station). Three more pints immediately postoperatively raised the over-all total to 24 pt. (Type O, 12 days of age) on the day of injury. As he recovered consciousness, his pressure rose spontaneously to 160/100, pulse 90 to 100, respirations 20 per minute. Fifteen hours postoperatively his pressure had fallen to 110/75, pulse 94, respiratory rate 20 per minute.

With the tourniquets in place, external blood loss seemed hardly to have exceeded 3000 cc. at the hospital. Postoperative bleeding was not noted.

Subsequently he was essentially afebrile, and his course was exceedingly smooth. Five hundred cubic centimeters of blood (fresh) was given on the second postoperative day and 1000 cc. on the third day. A secondary dressing, performed on the third postoperative day under spinal anesthesia, revealed his wounds to be clean. His course for the next two months was reported as uneventful.

Comment.—This patient also represents a response to massive wounds of the extremities. The steroid collection on the first day after injury was lost, and so the height reached during the first full 24-hour period is unknown. The peaks in the corticosteroid excretion on the 5th, 9th, and 10th days were inversely related to the eosinophile count (Fig. 6). These peaks cannot be clearly associated with the operative trauma and would appear to be independent of any known insult. This patient was one of the few casualties who demonstrated an increased excretion in 17-ketosteroids. Coincident studies of hepatic function demonstrated less hepatic impairment in this patient than in any seriously injured casualty studied.[8] This maintenance of normal hepatic

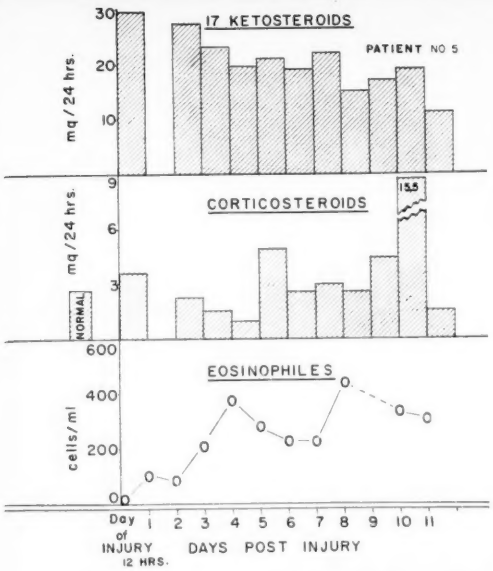
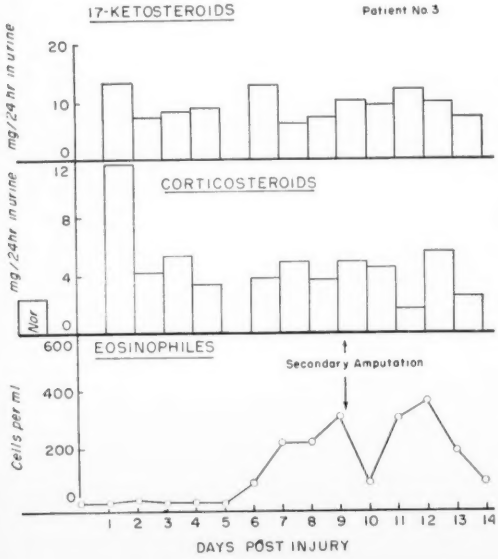


Fig. 6.—This patient had the most radical débridement, the healthiest wounds, the best hepatic function, and the greatest excretion of 17-ketosteroids of any casualty studied. These findings may represent additional evidence of the continuing effect of the wound on adrenal function.

function may account for the high 17-ketosteroid excretion. Conn [1] has suggested that the failure of the casualty to excrete an increased amount of 17-ketosteroids after severe trauma may be due to failure of the liver to form 17-ketosteroids from 17-hydroxysteroids. Those changes, when compared with those of other casualties, again suggest the importance of necrotic tissue and infection in explaining the marked changes seen in other patients.

Fig. 7.—This casualty had a continuing injury in the form of a gangrenous leg. Note the persistent absence of circulating eosinophiles and the persistent increase in the excretion of corticosteroids. These changes were more marked than in any other patient. The excretion of 17-ketosteroids was not increased.



CASUALTIES WITH WOUND INFECTION AND NECROSIS

PATIENT 3.—A 21-year-old American soldier, weight 170 lb., blood Type O, was in combat two months, in Korea four months.

This soldier stepped on a land mine near the Battalion Aide Station at 7 p. m., Aug. 3, 1952. His injuries included a perforation of the inferior vena cava, four perforations of the duodenum, eight perforations of jejunum and ileum, penetration of the chest, penetration of the left popliteal artery, shattering compound fractures of the left femur, left tibia, and right tibia, and multiple soft tissue wounds of both lower extremities.

After 200 cc. of albumin at the Aide Station, he was evacuated by helicopter, arriving one hour after injury. He was cold, pale, and restless, and the capillary refilling was slow. His blood pressure and pulse were unobtainable.

After 6 pt. of blood was given, his pressure was 100/60. After 9 pt. of blood was given, his pressure was 120/70, pulse 132.

Operation, lasting four hours, was begun five hours after injury. After a thiopental induction, ether and curare were utilized. The vena cava was ligated below the renal veins. The ileum was resected and multiple small bowel perforations were closed. Several soft tissue wounds were débrided. The popliteal artery was not bleeding externally, and because of the critical condition of the patient, the wound was not explored. For a short time during the operation, the blood pressure was unobtainable. For the last hour and one-half the pressure ranged between 80/50 and 100/60. By the end of operation he had been given a total of 21 pt. of Type O bank blood, averaging 15 days of age. Operative blood loss was measured at approximately 4000 cc. Postoperatively his pressure stabilized at 110/60 to 130/70 for 17 hours and then spontaneously rose to 160/90, pulse rate 130 per minute. Thereafter his pressure remained above 140/80 until his evacuation on his 14th postoperative day.

His postoperative course was febrile (101 to 102 F). Amputation below the left knee was necessary on the ninth day after injury because of gangrene subsequent to the perforation of the popliteal artery.

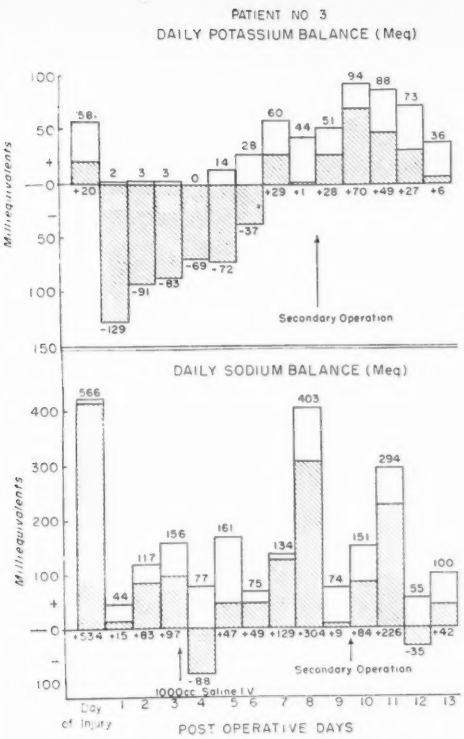


Fig. 8.—This patient with a gangrenous leg lost a tremendous amount of potassium during the first week. Amputation of the gangrenous limb on the ninth postoperative day did not result in a secondary loss of potassium but was associated with further conservation of sodium.

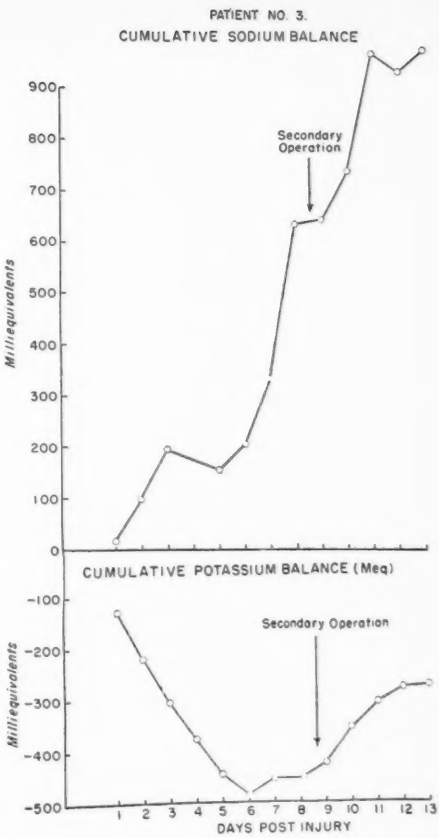


Fig. 9.—The conservation of sodium was progressive; the loss of potassium spontaneously reversed.

Comment.—This soldier had wounds which were almost fatal in extent. He developed gangrene of an extremity which was not amputated until the ninth day. Meanwhile, his corticosteroid excretion remained high and his eosinophile count was very slow to rise (Fig. 7). His eosinophile count fell transiently after the second operation. The 17-ketosteroid excretion was persistently low, among the lowest in the group studied. His potassium loss was the highest of any patient studied, reaching 489 mEq. (Figs. 8 and 9). The loss was extremely high for the first six days after injury (Fig. 8), and then the balance became positive at the time his eosinophile count started to rise. His sodium retention (Fig. 8) was also the most marked, reaching 970 mEq. (cumulative) by the 13th day (Fig. 9). Sodium retention occurred throughout the 13-day period.

This soldier demonstrates the changes associated with gangrenous tissue. He had gangrene of an entire lower limb with an associated infection. The result was a continued stimulation of the adrenal cortex and a continued loss of potassium and retention of sodium. The potassium loss may have represented cell destruction in the gangrenous limb. This gangrenous limb, owing to loss of cellular integrity, produced the equivalent of an increase in extracellular fluid space comparable to the volume of the gangrenous tissue. Thus in electrolyte equilibration potassium would be lost and sodium retained in the nonviable tissue (Fig. 10). Although his sodium retention was the most pronounced, it was not as striking, in comparison with older casualties, as was the potassium loss. It would appear that the

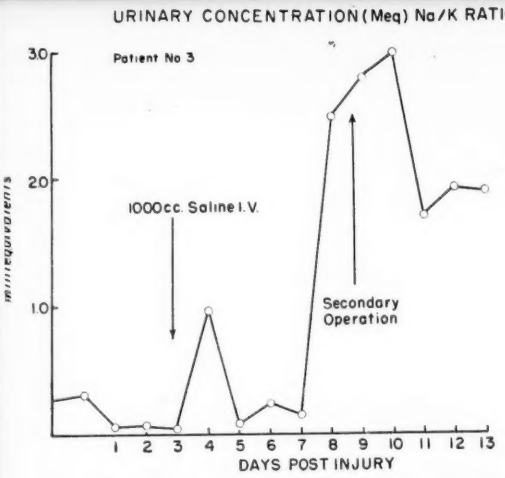


Fig. 10.—The patient excreted more potassium than sodium for seven days.

body not only separates dead tissue as a slough but may also have this mechanism which might be termed "metabolic débridement" by which products of tissue degeneration are absorbed, metabolized, or excreted. With the breakdown of cell membranes in a gangrenous limb, sodium and potassium must equilibrate throughout the total water of the extremity, and this must in turn equilibrate with the plasma, dependent upon its contact with the circulation. This phenomenon was best demonstrated in the casualties treated by the Surgical Research Team at the Renal Insufficiency Center in Korea. Some patients with post-traumatic renal insufficiency whose plasma potassium had been lowered by dialysis on the artificial kidney, later very rapidly developed a secondary rise in plasma potassium concentration. The investigators observing these patients learned to recognize this finding as evidence of hidden infection or necrosis.[9]

With injury, the patient's plasma sodium concentration fell to 128 mEq. in spite of sodium retention (Fig. 11). Thereafter, it rose slowly, only to fall again with the secondary operation. Coincident with the secondary operation, his plasma potassium rose as his sodium fell.

PATIENT 2.—A 21-year-old American soldier, weight 135 lb., blood Type B, was in combat two weeks, in Korea three and one-fourth months.

This soldier was wounded by fragments from a land mine at 2:30 a. m., July 18, 1952, while on patrol duty. His injuries included a traumatic amputation of the lower left leg and extensive soft tissue injuries of the right calf.

He arrived via litter and litter jeep at the Batallion Aide Station at 4:55 a. m. and via helicopter at the hospital at 6:30 a. m. No resuscitative measures were recorded during this time beyond control of hemorrhage. On admission to the hospital his blood pressure was 150/80, pulse rate 100. His pulse was of good quality, and his color was normal. He was quiet and alert.

Re-amputation of the left leg and débridement of the right leg were completed with the patient under spinal anesthesia by 8:35 a. m. During this time he received 850 cc. of Type O bank blood (22 days of age). Subsequent transfusions were of fresh,

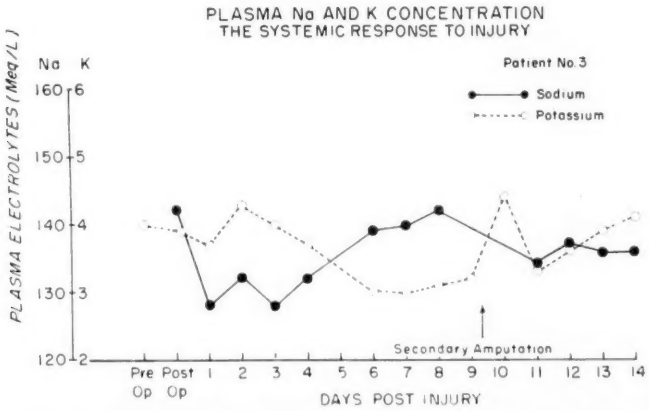


Fig. 11.—Fall in plasma sodium after each operation. The rise in plasma potassium, as seen here after the second operation, was often noted when determinations were made at frequent intervals.

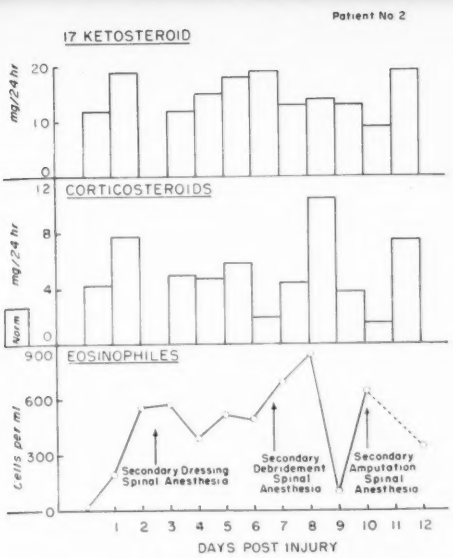
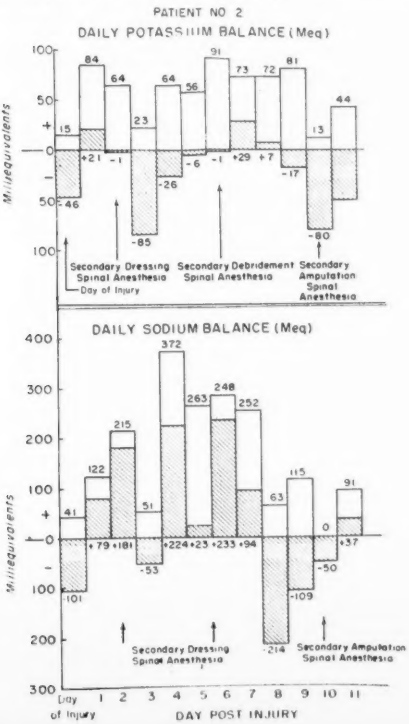


Fig. 12.—The effect of necrotic tissue in producing a continued stimulus to corticosteroid excretion. Repeated traumas produced delayed but repeated responses in corticosteroid excretion. Note the elevation in the eosinophile concentration to 840 cells per milliliter on the eighth day after injury.

type-specific blood, locally obtained. During operation his blood pressure ranged about 100/50, pulse 100, respirations 20 per minute. His operative blood loss, as measured by the washed sponge technique, was 450 cc. His postoperative course was marked by the development of fever on the third day. Spinal anesthesia was repeated for secondary dressings on the 2nd postoperative day, for re-débridement on the 6th postoperative day, and for re-amputation of the left leg on the 10th day. The re-amputation was necessary because of gangrenous muscle at the level of the previous amputation. Oral diet was repeatedly supplemented with intravenous fluids.

Fig. 13.—Secondary trauma tended to accentuate the response of the original injury.



Comment.—This case is primarily a study of repeated traumas. Three secondary operations were performed on the 2nd, 6th, and 10th days after injury. On the second day after injury, the original adrenal response was still manifested, and no further response was detectable. After the latter two operations, a sharp response occurred in each instance.

This soldier is one of two studied who had residual necrotic tissue. The necrotic tissue in the wounds of this casualty was not extensive. His eosinophile count fell to zero (Fig. 12) and then rapidly rose, finally reaching the very high level of 840 on the eighth day. Following secondary débridement on the sixth day, there was a late fall coincident with a secondary rise in corticosteroid excretion. The corticosteroid excretion remained elevated throughout five days of study prior to the secondary débridement of the wounds. It then fell to normal but again became elevated after secondary operations on the 6th and 10th days. The 17-ketosteroid excretion was not elevated.

Daily balance studies (Fig. 13) demonstrated a tendency to conserve sodium until the eighth day after injury. He then developed a negative sodium balance which was reversed by secondary trauma. This reversal, following secondary trauma on the 10th day, was coincident with a sharp decrease in the excretion of water (Fig. 17), which had roughly paralleled his sodium balance. His cumulative sodium balance is indicated in Figure 14. Potassium loss followed the primary injury and the secondary injuries on the 2nd and 10th days (Fig. 13). His cumulative potassium loss was therefore increasing at the time of his evacuation on the 12th day (Fig. 14). Figure 15 demonstrates a very sharp decrease in the ratio of

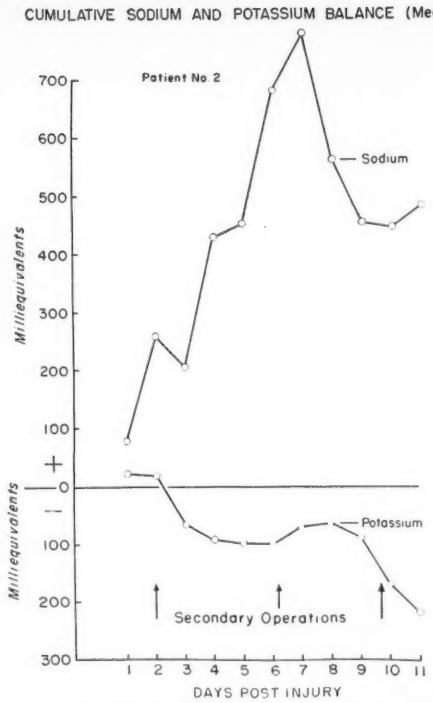
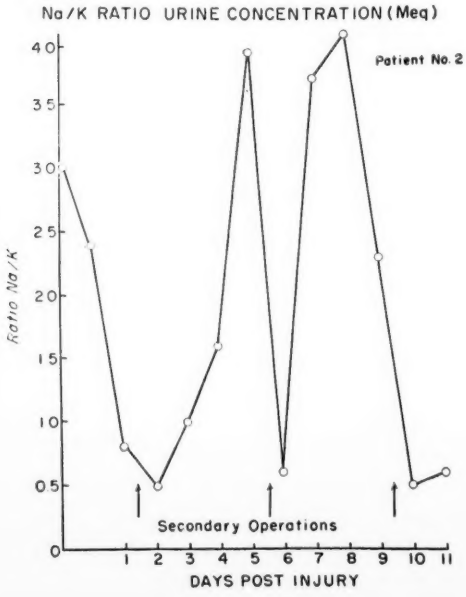


Fig. 14.—The loss of potassium was continuing at the end of 11 days.

Na : K excretion following each of the three secondary insults. The consistent inverse changes in the plasma sodium and potassium concentration were again found after each

Fig. 15.—The effect of repeated traumas on the urinary excretion of electrolytes. The first two points represent preoperative and postoperative values.



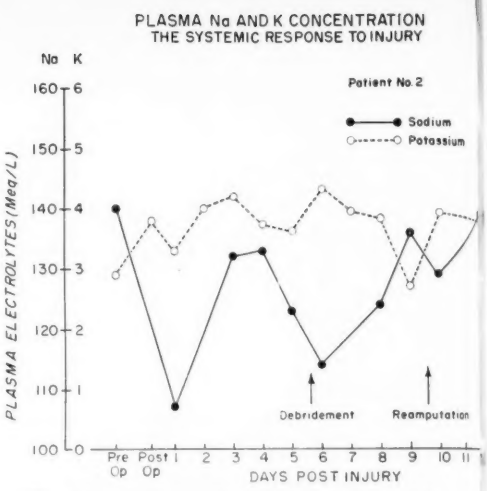
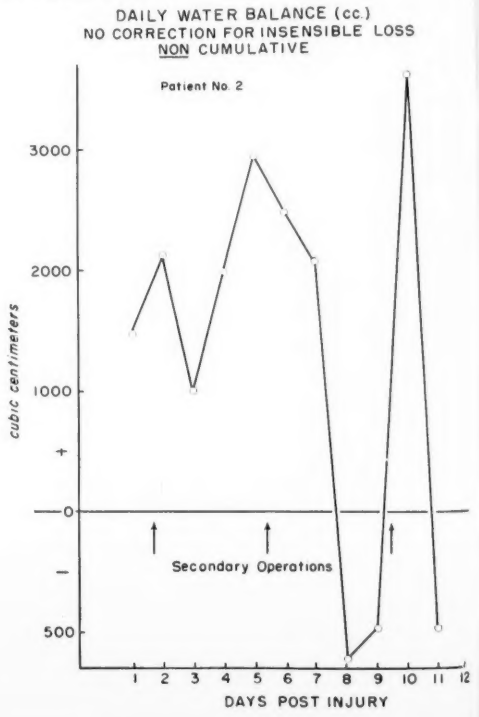


Fig. 16.—Three operations were each followed by a marked fall in plasma sodium concentration and a slight rise in potassium concentration.

injury (Fig. 16). Secondary trauma resulted in a sharp fall in plasma sodium and a slight rise in plasma potassium concentration.

Patient 16.—A 20-year-old American soldier, blood Type B, was wounded at 1 p. m., Sept. 3, 1952, by the discharge of a carbine at close quarters. The bullet perforated the liver, stomach, transverse colon, diaphragm, and left lung.

Fig. 17.—Secondary operation on the 10th day reversed the diuresis.



At the Battallion Aide Station he was given 100 cc. albumin, 600,000 units of penicillin, and tetanus toxoid. At 2:20 p. m. he was given 500 cc. of blood at the Collecting Station.

When he arrived at the hospital at 6:40 p. m., his blood pressure was unobtainable. He was pale, dry, and very quiet. His pulse was barely perceptible at the wrist. His veins were not visible, and capillary refilling was slow.

After receiving 1500 cc. of blood in 15 minutes, his pressure was 90/40. After 3000 cc. of blood, part intra-arterially, his pressure was 125/75. As did many other patients, he complained of cold and shook the litter by shivering.

Operation was started at 7:40 p. m. His pressure was 170/80, pulse rate 125. After 3500 cc. of blood had been given at the hospital, operation was begun under thiopental (300 mg. total), ether, oxygen, and curare (80 units total). His pressure gradually dropped to 90/50, pulse rate 140 per minute.

Operation revealed 300 cc. of blood in the peritoneal cavity. Another 500 cc. was removed during the course of the operation. Finally 1000 cc. was removed from the left pleural cavity. Operation consisted of drainage of the liver, colostomy, and closure of the gastric wounds. The pleural cavity was aspirated, and the wounds of entrance and exit were débrided. An additional 2000 cc. of blood (6000 cc. total, Type O, 12 days of age) was given during operation. His pressure ranged between 90/60 and 105/60 most of the two hours required for operation.

His postoperative course was marked by hemoconcentration with an hematocrit value reaching 63% for a few hours. His oral temperature ranged from 101 to 103 F secondary to a wound infection.

Comment.—This casualty demonstrates the continued adrenal response to the continued trauma of infected wounds and the repeated traumas of thoracentesis. He remained febrile throughout the 12 days of study. His corticosteroid excretion remained high during most of his hospital course (Fig. 18). His eosinophile count was slow to rise. Again, he demonstrated minor changes in plasma sodium and potassium concentrations after the secondary minor injuries of repeated thoracentesis under local anesthesia.

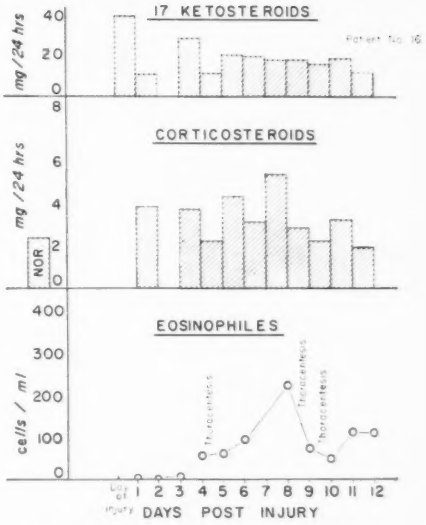


Fig. 18.—17-Ketosteroid excretion was increased on the day of injury in spite of an hepatic wound. Corticosteroid excretion remained high owing to the continued trauma of infection and the repeated trauma of thoracentesis. The eosinophile concentration was slow to rise.

SUMMARY AND CONCLUSIONS

A study of adrenal function was performed on 20 critically injured battle casualties during the first 7 to 14 days after injury. The studies demonstrate the following points:

1. Under the conditions of battle in Korea during 1952, the stress of combat did not lead to adrenal cortex exhaustion. Each casualty studied demonstrated the adrenal cortex response to trauma.

2. The corticosteroid (formaldehydogenic compounds) excretion rose sharply following trauma. The maximal excretion was sometimes not reached until the second day after injury. During an uncomplicated postoperative course, the corticosteroid excretion fell to normal about the third or fourth day after injury and then often demonstrated a slight secondary rise.

3. The 24-hour excretion of 17-ketosteroids was usually normal. A few patients demonstrated a slight rise. The most marked elevation in excretion occurred in a patient who had the most radical débridement, the healthiest wounds, and the best hepatic function.

4. The concentration of circulating eosinophiles invariably fell to near zero after injury. In the uncomplicated case, a return to normal occurred a few days later. A rebound increase above normal sometimes followed before the concentration stabilized at a normal level.

5. During the first 24 hours after trauma, the plasma sodium concentration decreased and the plasma potassium concentration rose. The latter phenomenon lasted only a few hours.

6. With injury, the retention of sodium and excretion of potassium characteristically resulted in the excretion of more potassium than sodium. This was best demonstrated by expressing the urinary concentration of the two electrolytes (milliequivalents) as a ratio of sodium to potassium. With injury this ratio fell to a range of 0.5 to 0.1.

7. Balance studies demonstrated the tremendous and prolonged retention of sodium and loss of potassium which occurred in some of these patients.

8. Repeated traumas resulted in repeated responses of the adrenal cortex. If the secondary trauma occurred during the primary adrenal response, no further increment could be recognized. Subsequently, secondary trauma was characteristically followed by a secondary rise in corticosteroid excretion and in plasma potassium concentration and excretion and by a secondary fall in plasma sodium concentration and often by a further sodium conservation. The secondary fall in eosinophile concentration was limited in extent and lasted only a few hours.

9. Wound infection and necrosis were associated with a continued adrenal cortex and electrolytic response of a magnitude and duration not seen in other casualties. The loss of such large quantities of potassium suggests also a process of "metabolic débridement" of necrotic tissue.

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Experimental Vascular Grafts

VII. Effects of Growth on Pericardial Autografts

RALPH K. ZECH, M.D.
LLOYD M. NYHUS, M.D.
CHARLES A. GRIFFITH, M.D.
and
HENRY N. HARKINS, M.D., Seattle

Recent advances in the procurement and processing of arterial substitutes have greatly extended the applicability of vascular surgical procedures. Valid criticisms, however, may be brought forward for each of the methods, whether the new prosthetic device be arterial homograft, arterial heterograft, or plastic mesh tube. Continued search for an ideal vascular replacement is thus indicated.

This report relates our experiences with autogenous pericardial grafts implanted into the thoracic aortas of growing pigs. An attempt has been made to evaluate the effects of rapid growth (with its attendant stresses) upon these pericardial autografts. Fascial supported and nonsupported pericardial autografts were studied.

Sako * originally studied pericardial autografts implanted into the thoracic aortas of adult dogs. He reported that under the conditions of his experiments, the pericardial transplants tended to dilate, but the dilatation could be prevented by a perigraft wrap of fascia lata. It is also of particular interest that these autogenous grafts were generally free of severe degenerative change, although some cholesterol and calcific deposition was noted.

PROCEDURE

Ten weanling pigs, averaging 19 kg. in weight, were operated upon. In each animal a sheet of pericardium averaging 6 × 8 cm. was excised and fashioned into a tube over an 0.8-cm. Lucite shunt. Carrell over-and-over continuous nonabsorbable 00000 (silk) sutures were used throughout. (The longitudinal suture was reinforced by two or three interrupted mattress sutures.) The grafts were then sutured into a defect made in the thoracic aorta. Five animals were given nonsupported grafts, while five others were fitted with a pericardial graft which was then encased in a sheet of fascial tissue removed from the central tendinous portion of the diaphragm. This fascial sleeve was sutured so as to cover the entire graft and 0.5 to 0.7 cm. of adjacent aorta. The defect in the diaphragm was repaired with absorbable (gut) sutures.

RESULTS

Seven pigs reached maturity, while three did not. Three of the surviving animals had nonsupported grafts, while the remainder had supported pericardial grafts. Two of the animals which died prematurely were of the nonsupported series; one of these died of graft rupture one month postoperatively, and one died of thrombosis of the graft four months after implantation. One animal having a supported graft died of a hemorrhage between the inner pericardium and outer fascial sheath that caused an obliteration of the graft lumen.

All pericardial grafts removed at slaughter were patent and functioning well. The three nonsupported grafts were thin-walled, and definite aneurysmal dilatations were present in two while the third was diffusely enlarged and had a dangerously thinned area 1 cm. from the lower anastomosis (Fig. 1). The grafts were all tough and inelastic. The in-

From the Department of Surgery, University of Washington School of Medicine. This work was supported by Grant H-1136, National Institutes of Health.

Cardiovascular Trainee, National Heart Institute (Dr. Zech); Postdoctoral Research Fellow, National Institutes of Health (Dr. Nyhus); Postdoctoral Research Fellow, National Institutes of Health (Dr. Griffith), and Professor of Surgery, University of Washington School of Medicine (Dr. Harkins).

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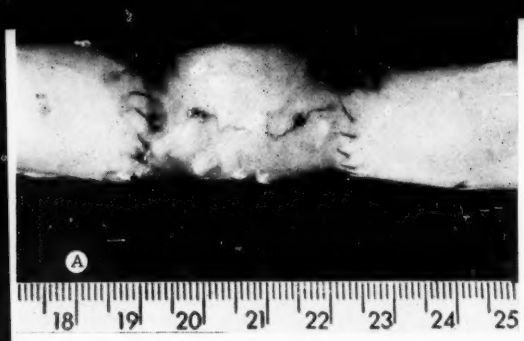


Fig. 1.—Results in a nonsupported pericardial autograft of the thoracic aorta (Pig 175).

A, inflated view (120 mm. Hg): Note the fusiform swelling on the anterior surface of the graft. Also note the straightening of the longitudinal continuous suture which occurred with growth. The interrupted mattress sutures used to reinforce the longitudinal continuous suture are also seen.

B, open view: Note the areas of thinning adjacent to the distal anastomosis, also the subintimal plaques. Microscopically, the intima was of variable thickness.

tima was heavy and generally smooth, although there were several subintimal plaques. The intima was unusually heavy over the sutured areas. There were no mural thrombi in the aneurysmal sacs, and the intima lining of each aneurysm was smooth, glistening, and gray.

The supported pericardial grafts were similar to nonsupported grafts in almost every respect (Fig. 2). There was, however,

Growth Changes in Pericardial Autografts: Supported and Nonsupported

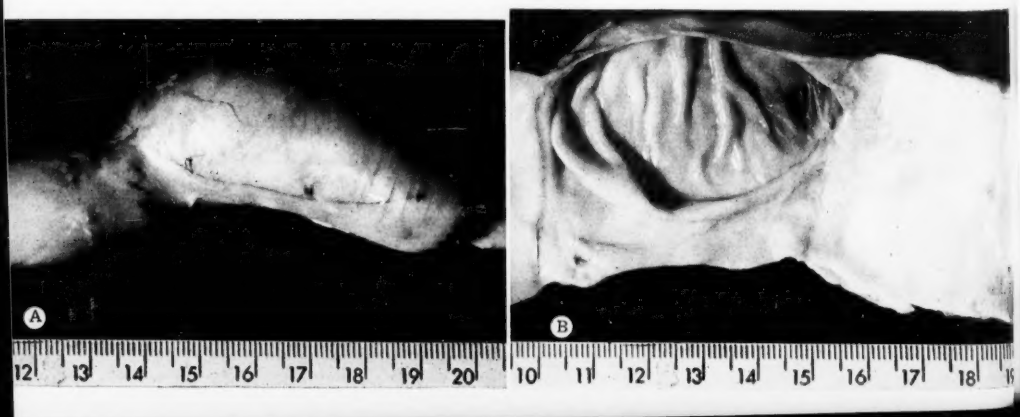
Pig No.	Type of Graft	Weight Increase, %	Linear Crown-rump Growth of Animal, %	Linear Growth, %		Diameter Increase, %	
				Aorta	Graft	Aorta	Graft
168	Supported	510	75	70	45	60	60
174	Supported	600	102	55	54	100	200
178	Supported	420	50	95	71	95	170
172	Supported	670	110	107	—7	80	40
175	Nonsupported	500	60	100	93	60	120
176	Nonsupported	560	60	80	51	75	90
182	Nonsupported	500	72	100	10	70	95
Average		537	76	87	45	77	111

Fig. 2.—Results in a supported pericardial autograft of the thoracic aorta (Pig 174).

A, inflated view (120 mm. Hg): Note the pronounced dilatation of the graft as well as the fraying of the fascial buttress. In this instance growth was accompanied by maximal straightening of the longitudinal suture and angulation of the graft.

B, open view: Note the thick, smooth intima. The large aneurysm is dangerously thinned near the lower anastomosis. The fascial sheath can be identified adjacent to the upper anastomosis at the upper left hand side of the figure.

one exception; this was a supported pericardial graft placed in Pig 172 (Table) which exhibited minimal growth and prominent degenerative changes. No definite cause could be found for these findings in this one supported graft. The remaining grafts possessed aneurysms, generally saccular. The intima was smooth, glistening, and gray, and it was especially heavy over the suture lines.



The walls of the supported grafts were thicker than those of the nonsupported grafts, but this thickening was caused by a fusion of pericardium to the fascial sheath. The encircling fascia-like tendon was often thinned. This was especially true in the grafts with aneurysmal dilatations. At times the attenuation was so marked that the fibers of the fascial buttress could not be identified.

With one exception (Pig 172), linear "growth" occurred in all grafts, with about an equal rate for both the supported and nonsupported types. However, this growth was less rapid than it was in the adjacent aorta (Table). Graft diameters increased at a greater rate than did the adjacent aorta, and it appears that the nonsupported grafts were less prone to such enlargement than were the supported grafts.

Microscopically, all grafts, except that in Pig 172, presented a similar picture. The supported grafts had a rather thin layer of acellular fibrous tissue with some areas of hyaline tissue interspersed. This tissue was joined to the outer layer of pericardium by a blending of tissue structures and acellular scar tissue. The pericardium itself was lined by an endothelium of variable thickness. Pericardial cellular structure was discernible, and there were areas of hyaline degeneration, amorphous depositions, and some subintimal calcification. Collagen was abundantly deposited in all pericardial grafts, and cellularity was reduced. There were no signs of intramural inflammatory reaction present, except about the strands of suture. The exception to the above description, in Pig 172, revealed a mass of calcification with minimal histologic evidence of pericardial or fascial tissues having once been present. Representative sections of a typical nonsupported and supported pericardial graft are seen in Figure 3.

Fig. 3.—Microscopic sections from pericardial autografts of the thoracic aorta.

A, nonsupported (Pig 175). The pericardial graft is to the left, the suture line (with silk suture in cross section above) in the middle, and the aorta to the right. The vessel lumen is below. There is a small amount of adventitia above to the right. The elastic tissue in the aorta is readily visible. Weigert's stain.

B, supported (Pig 174). The pericardium is below, and the supporting diaphragmatic tissue is above. The pericardial tissue is uniform in thickness but relatively acellular. Hematoxylin and e. Stain. The dark streak near the left edge of this picture is an artifact.

COMMENT

The pericardial autografts of this series show evidence of degenerative change and dilatation which are both more frequent and severe than reported by Sako.[3] These changes, with the exception of the aneurysms and the constriction in Pig 172 are not severe when compared with changes we frequently have found in arterial homografts similarly implanted.†

The grafts of this series have not been subjected to as prolonged implantation as have Sako's,[2] the average age of the grafts of this series being seven months; however, our animals underwent rapid growth. This appears to subject grafts, especially those in the thoracic aorta, to a maximal stress. The pericardial grafts implanted by Sako into the thoracic aortas of adult dogs probably were not subjected to the added factors of increasing intravascular pressures with age and other stresses of rapid "growth."

Dilatation was more prominent in both the supported and nonsupported pericardial transplants in our experiments than similar type transplants reported by Sako. A possible

† References 4 and 5.

explanation for this is that the pericardium of a weanling pig may not be as strong as that of the adult dog. Another variable certainly was added when it became necessary in the weanling pig to utilize the central tendon of the diaphragm as a fascial support for the pericardial autografts. There is no available source of fascia in small pigs such as the fascia lata of adult dogs. The gross appearance and tensile strength of the diaphragmatic tendon of the weanling pig used for buttressing these transplants of pericardium was comparable to fascia lata of the adult dog. Puppies might have been used for these experiments, but the advantage of as rapid growth would have been lost.

It is difficult to explain why dilatation of the grafts supported by fascia was greater than in the nonsupported animals. This may be related to an initial period of suppressed nutrition of the pericardium and tendon because each was separated from a source of such nutrition, namely, the tissue fluids in the case of the pericardium, and the blood stream in the case of the fascial encasement.

In general, degenerative changes exclusive of aneurysms were less frequently encountered in the pericardial grafts than they are in homologous transplants of artery.‡ The frequent occurrence of aneurysmal dilatations would appear to more than offset any relative advantage in lack of degenerative change that these grafts would have in comparison with arterial homografts.

SUMMARY AND CONCLUSIONS

Pericardial autografts implanted into the thoracic aorta of a growing pig tend to dilate and undergo frequent aneurysmal formation in spite of fascial support.

Longitudinal "growth" occurs in pericardial autografts, but it does so at a reduced rate.

Degenerative changes occur in pericardial autografts of the thoracic aorta but they tend to be less severe than those seen with homologous arterial grafts.

Pericardium as an arterial graft would not seem ideally suitable for implantation in patients on the basis of these experiments performed in growing pigs.

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Uropepsin as a Measure of Gastric Secretion

STANLEY P. RIGLER, M.D.
HARRY A. OBERHELMAN, M.D.
MAUDE M. HANKE, B.A.
and
LESTER R. DRAGSTEDT, M.D., Chicago

The identification and measurement of uropepsin, a substance found in the urine which is thought to be identical with pepsinogen, has prompted the idea that uropepsin determinations may be a simple and accurate index of gastric secretory activity.

Available evidence indicates that pepsinogen present in gastric glands is secreted by two routes: directly into the lumen of the stomach, where it is converted to pepsin, and into the blood stream, whereby it is carried to the kidneys and excreted as uropepsin.*

Gray and co-workers have demonstrated the importance of the hypothalamic-pituitary-adrenal axis in the control of uropepsin excretion.† Administration of corticotropin (ACTH), cortisone, or hydrocortisone (Compound F) to patients produces an average increase in uropepsin excretion of almost 200%, paralleled by an equal increase in gastric pepsin and HCl output. That this increase is mediated by the adrenals is shown by the fact that patients with Addison's disease have very low levels of uropepsin and that administration of cortisone increases their uropepsin excretion but corticotropin does not. Similarly, patients with adrenal hyperfunction (Cushing's disease) and hypothalamic tumors have abnormally elevated uropepsin levels.[5]

HCl OUTPUT AND DUODENAL ULCER

Both the volume and free acidity of gastric juice are elevated in patients with duodenal ulcer.[6] Zubiran and others have shown that administration of corticotropin or cortisone to dogs results in a 20%-50% increase in HCl output from stomach pouches and that this increase is independent of vagal innervation or the presence of the gastric antrum.[7] Gray has demonstrated the same phenomenon in humans.

GASTRIC PEPSIN AND DUODENAL ULCER

Gastric pepsin secretion appears to parallel HCl output. Measurement of basal, unstimulated gastric secretion in duodenal ulcer patients demonstrates pepsin levels above three times that of normal.[8] The importance of the vagus in pepsin secretion is attested to by the high pepsin content of insulin-stimulated gastric juice as opposed to histamine juice. Further evidence is the observation of Dragstedt and others (unpublished data) that dogs with innervated pouches secrete 5 to 10 times as much pepsin as do dogs with denervated pouches, both in terms of concentration and total output.

UROPEPSIN AND DUODENAL ULCER

Gray and others have shown that patients with duodenal ulcer have uropepsin levels two to three times that of normal persons.‡

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From the Department of Surgery of the University of Chicago.

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* References 1 and 2.

† References 3 and 4.

‡ References 4 and 5.

This observation, as well as the data related to the importance of the adrenals in regulating uropepsin excretion, has led him to suggest that peptic ulcer may be a result of a hypersecretion of gastric juice brought about in part by increased adrenal cortex activity.

It appears to us that two very important questions have been raised by the observations mentioned. 1. Does uropepsin excretion accurately reflect the secretory activity of the stomach? 2. Do the variations in uropepsin levels suggest that stress mediated by the adrenal cortex is a significant factor in the pathogenesis of duodenal ulcer?

MATERIAL

This paper reports the results of our experience with uropepsin determinations in three groups of patients. The first consists of patients admitted to the hospital with diagnoses other than peptic ulcer. The second is comprised of patients studied prior to undergoing surgery for what subsequently proved to be duodenal ulcer. The third group is composed of patients who had vagotomy with or without gastroenterostomy for duodenal ulcer six months to eight years previously. This group was studied when they returned to the hospital for routine follow-up secretory studies. All were free of evidence of recurrence at the time studied.

METHODS

Nocturnal gastric secretions were determined as previously described.[6] The volume was measured, the free acid determined by titrating against Toepfer's reagent, and the output of HCl calculated in milliequivalents by multiplying the volume, expressed in liters, by the free acid, expressed in clinical units.

Concurrently, peptic activity in gastric juice and urine was determined as follows. A 1:50 dilution of gastric juice was made with 0.06 N HCl, giving a a solution of pH 1.2=1.3. One cubic centimeter of this diluted sample was added to 5 cc. of 2% acidified hemoglobin and incubated for 10 minutes at 25 C. The reaction was stopped by adding 10 cc. of 5% trichloroacetic acid. The concentration of tyrosine-like substance in 5 cc. of filtrate was determined by the Folin-Ciolcatu procedure.

Concurrent 12-hour urine specimens were collected and refrigerated. A 10 cc. aliquot was taken and adjusted to pH 1.2=1.5 with 0.3 N HCl; 0.2 cc. of this acidified urine was made up to 1 cc. with 0.06 N HCl. This sample was incubated at 25 C with 5 cc of 2% acidified hemoglobin for 10 minutes. At the end of this time 10 cc. of 5% trichloroacetic acid was added to stop the reaction. The concentration of tyrosine-like substances in 5 cc. of filtrate was determined.

The chromogenic substances present in urine and gastric juice were corrected by preparing blank tubes containing 1 cc. of diluted urine or gastric juice, 10 cc. of trichloroacetic acid, and 5 cc. of hemoglobin. The difference between the chromogenic substances in the sample and in the blanks was determined and plotted against a standard curve.

One unit of peptic activity is arbitrarily defined as that amount of enzyme which will release the color equivalent of 0.1 micromole of tyrosine-like substance from 0.1 gm. of hemoglobin substrate in 10 minutes. Results are expressed in peptic units per hour.

RESULTS

Sixteen patients with diseases other than gastric or duodenal ulcer had a mean HCl output of 16 mEq., a mean gastric pepsin secretion of 29,706 peptic units per hour, and

TABLE 1.—*Non-Ulcer Patients*

Patients, No.	Mean HCl Output, mEq.	S. E.	Mean Gastric Pepsin, P. U./Hr.	S. E.	Mean Uropepsin, P. U./Hr.	S. E.
16	16	±1.8	29,706	±5,436	142	±36

TABLE 2.—*Duodenal Ulcer Patients*

Patients, No.	Mean HCl Output, mEq.	S. E.	Mean Gastric Pepsin, P. U./Hr.	S. E.	Mean Uropepsin, P. U./Hr.	S. E.
29	48	±4.7	62,824	±6,331	146	±23

TABLE 3.—*Duodenal Ulcer Patients: Relationship of Gastric Pepsin to Acidity*

	Patients, No.	HCl Output, mEq.
Gastric pepsin less than 50,000 P. U./hr.	15	22
Gastric pepsin more than 50,000 P. U./hr.	14	65

a mean uropepsin excretion of 142 peptic units per hour (Table 1).

The series of duodenal ulcer patients studied preoperatively had an average HCl output of 48 mEq., an average of 62,824 peptic units per hour of gastric pepsin, and a uropepsin excretion of 146 peptic units per hour (Table 2). Five of our duodenal ulcer patients excreted no uropepsin, while 5 excreted from 1 to 50 peptic units per hour, and 19 had levels of over 50 peptic units per hour.

TABLE 4.—*Duodenal Ulcer Patients: Relationship of Uropepsin to Gastric Pepsin and Acidity*

	Patients, No.	HCl Output, mEq.	Gastric Pepsin, P. U./Hr.
No uropepsin	5	38	40,298
Uropepsin 0-50	5	37	48,867
Uropepsin over 50	19	50	68,886

In general, HCl output and gastric pepsin varied directly in these patients, those with highest gastric pepsin levels having the highest outputs of HCl (Table 3). The relationship of uropepsin excretion to the other two quantities was less obvious. Five patients with no uropepsin had an average HCl output of 38 mEq. and a mean gastric pepsin of 40,298 peptic units per hour, while five patients averaging 1 to 50 units of uropepsin per hour had a mean gastric pepsin secretion of 41,867 peptic units per hour and a mean HCl output of 37 mEq. (Table 4). Studies on 23 patients who had vagotomy alone or combined with gastroenterostomy for duodenal ulcer six months to eight years previously revealed a mean HCl output of 7.6 mEq., an average gastric pepsin value of 19,047 units, and a mean uropepsin excretion of 97 peptic units per hour (Table 5).

TABLE 6.—*Postvagotomy Patients: Relationship of Gastric Pepsin to Acidity*

Gastric Pepsin, P. U./Hr.	Patients, No.	HCl Output, mEq.
300-10,900	5	0
10,000-30,000	12	8
30,000 and above	6	16.3

The relationship of gastric pepsin to HCl output again appears to be fairly direct (Table 6). But that of uropepsin to the other two measured quantities appears, if anything, to be inverse. As was seen previously, patients with no detectable uropepsin have appreciable quantities of gastric pepsin and HCl (Table 7).

TABLE 7.—*Postvagotomy Patients: Relationship of Uropepsin to Gastric Pepsin and Acidity*

	Patients, No.	HCl Output, mEq.	Gastric Pepsin, P. U./Hr.
No uropepsin	7	14	31,000
Uropepsin over 100	9	7	20,732

Comparison of the two groups of duodenal ulcer patients, one studied preoperatively and the other from six months to eight years postoperatively, reveals a reduction in HCl out-

TABLE 5.—*Duodenal Ulcer Patients—Postvagotomy*

Patients, No.	Mean HCl Output, mEq.	S. E.	Mean Gastric Pepsin, P. U./Hr.	S. E.	Mean Uropepsin, P. U./Hr.	S. E.
23	7.6	±2.5	19,047	±2,890	97	±21

put of 88%, a reduction in gastric pepsin levels of approximately 66%, and a reduction in uropepsin levels of approximately 30%.

A striking observation noted by others is the profound effect of surgery on uropepsin excretion. The highest values we have observed have been in patients during the first three or four days after vagotomy. The typical patient excretes from 500 to 1500 peptic units per hour in the immediate postoperative period. During the ensuing days the uropepsin level gradually falls.

COMMENT

Examination of the variations between HCl output, gastric pepsin secretion, and uropepsin levels in the same group of patients reveals a rather complex interrelationship. Gray has shown that administration of adrenal steroids produces a rise of equal magnitude in all three values, but it is not surprising that such pharmacological alterations are not completely borne out in clinical studies.

The parallel relationship of gastric pepsin and HCl output described many times in the past is confirmed in these studies. But uropepsin does not seem as clearly linked with either HCl output or gastric pepsin as has been previously suggested. Thus, in our series of duodenal ulcer patients studied preoperatively, five patients with no uropepsin exhibited an average HCl output which was more than twice normal. Further, comparison of duodenal ulcer patients studied preoperatively with those observed following vagotomy reveals that, while HCl output and gastric pepsin secretion are reduced below normal, uropepsin excretion remains elevated.

Thus, in our experience uropepsin levels are independent of gastric secretion activity as measured by HCl output and by gastric pepsin secretion.

Abundant proof exists that uropepsin excretion is intimately related to adrenal cortex function, indeed, that uropepsin may be a sensitive and accurate index of adrenal activity. Seemingly, any situation of stress, physical or emotional, acute or chronic, results in an elevation of uropepsin level.

The demonstration that duodenal ulcer patients have abnormally high uropepsin levels and that the administration of adrenal corticoids produces increased uropepsin excretion are most striking and interesting phenomena. These studies, in addition to the well-documented evidence that administration of large doses of adrenal corticoids have caused exacerbation of ulcer symptoms or the appearance of duodenal ulcers not previously demonstrated, suggest that an alternate mechanism for the production of a hypersecretion of gastric juice does exist. That this mechanism can be brought into play under certain specific circumstances, such as those mentioned above, seems clear. But this does not warrant the assumption that this alternate mechanism actually does play a part in the pathogenesis of peptic ulceration except in those very specific instances.

We believe that peptic ulceration is a result of a hypersecretion of gastric juice of vagal origin. This hypersecretion is almost invariably demonstrable in duodenal ulcer patients. Cutting the vagi eliminates the hypersecretion and permits the ulcer to heal. We believe that this is a simple and clear-cut experiment which effectively demonstrates the pathogenesis of the disease.

SUMMARY

Patients with duodenal ulcer have elevated HCl output and gastric pepsin secretion. However, their uropepsin excretion is not significantly elevated.

Gastric pepsin and HCl output appear to vary directly. Uropepsin does not vary consistently with the other two quantities.

Some duodenal ulcer patients exhibited elevated HCl and gastric pepsin levels while excreting no uropepsin.

Patients who had vagotomy for duodenal ulcer have HCl and gastric pepsin levels be-

low that of non-ulcer patients. Their uropepsin levels are likewise reduced.

Markedly elevated uropepsin excretion is observed in the immediate postoperative period.

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Traumatic Cholecystocutaneous Fistula

ORVILLE F. GRIMES, M.D.
and
HOWARD L. STEINBACH, M.D., San Francisco

Perforation of the gall bladder into an adjacent viscus, the peritoneal cavity or the abdominal wall incident to cholelithiasis or cholecystitis is well known. It is quite uncommon, however, for fistulae to develop between the gall bladder and the anterior abdominal wall as a result of trauma to the gall bladder without associated injury to adjacent structures. The following report presents the details of an unusual case in which traumatic rupture of the gall bladder resulted in a cholecystocutaneous fistula and deposition of a gallstone which eventually became encysted within the abdominal wall.

REPORT OF A CASE

A 70-year-old white housewife fell down a flight of stairs four months prior to her hospital entry, causing marked contusion of the musculature of the right upper abdomen and a fracture of the right wrist. During the subsequent two weeks, she experienced mild pain in the upper abdomen, which finally localized in the right epigastrium. A tender subcutaneous mass gradually became evident. The patient consulted her local physician, who incised the mass under local anesthesia to allow drainage of a large accumulation of greenish fluid under considerable pressure. The wound continued to discharge copious amounts of bile, necessitating numerous daily changes of dressings for the next six weeks.

At physical examination upon hospital entry, the pertinent findings were limited to the abdomen, and particularly to the right upper quadrant. An opening, 0.5 cm. in diameter was present 4 cm. to the right of the midline about midway between the right costal margin and the umbilicus. A free flow of bile was noted through the orifice, and the skin surrounding this area was slightly irritated. The patient stated that on occasion particulate material exuded from the opening. However, no food particles had been noted in the discharge.

X-ray studies were performed by injecting 30% iodopyracet (Diodrast) through a catheter inserted into the fistulous opening. The opaque medium flowed through the fistula into the fundus of the gall bladder, which appeared to be adherent to the anterior abdominal wall. A large radiolucent filling defect was present at the tip of the fundus and was thought to represent an impacted gallstone. From the gall bladder, the opaque medium passed into the cystic and common ducts and then into a markedly distended duodenal loop (Figs. 1*a* and 1*b*).

Because the fistula persisted over a 4-month period without decrease in drainage and because of the possibility of recurrent bouts of infection, surgical excision of the tract and the gall bladder was deemed necessary.

The abdomen was opened through a right rectus muscle-splitting incision independent of and lateral to the opening of the fistula onto the abdominal wall. Considerable induration of the musculature of the right upper quadrant was noted as the incision was developed through these layers. The omentum was adherent to the anterior peritoneum over a wide area and had effectively walled off the fistulous tract from the remainder of the abdominal viscera. The peritoneum was markedly thickened and edematous in the right upper quadrant, suggesting both acute and chronic inflammation.

The fistulous tract was identified from the skin surface of the abdomen downward through the abdominal wall by means of a flexible probe. Within the peritoneal cavity, it communicated directly with the fundus of the gall bladder (Fig. 2). The gall bladder itself was definitely diseased, and its wall was thickened and partially fibrotic. Two rounded 0.5-cm. stones were noted within its lumen. The common bile duct was slightly enlarged but contained no stones. The head of the pancreas was only slightly indurated. Although the duodenum was dilated, there was no evident involvement of this segment of bowel or of the transverse colon. The fistulous channel in the abdominal wall was excised elliptically and allowed to drop into the peritoneal cavity. The entire tract was then excised in continuity with the gall bladder.

During the operative procedure the marked thickening about the fistulous channel in the abdominal wall was thought to be due merely to the

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From the Departments of Surgery and Radiology, University of California School of Medicine.

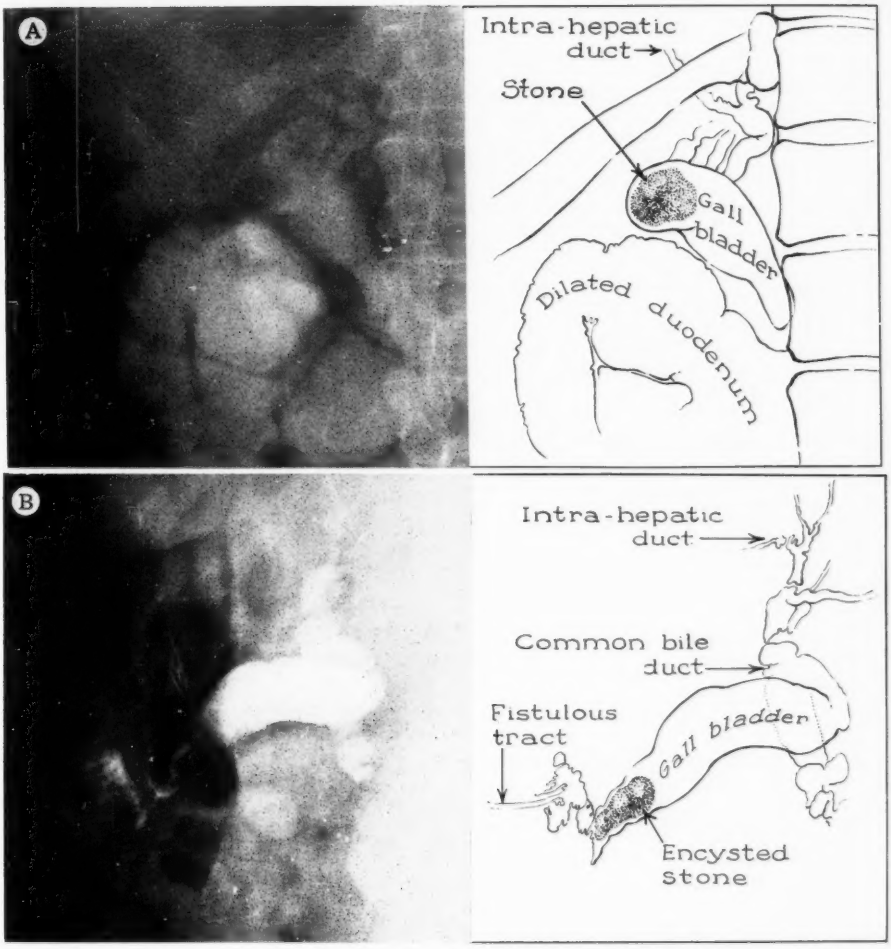

Fig. 1.—(*a*) Anteroposterior roentgenogram of the right upper quadrant following injection of iodopyracet (Diodrast) into the fistulous tract. The stone can be seen as a radiolucent shadow projecting into the lumen of the fundus of the gall bladder. The common bile duct is patent, allowing the opaque medium to pass into a dilated duodenal loop.

Fig. 1.—(*b*) Right lateral roentgenograms demonstrating the fistulous tract and the encysted stone in the abdominal wall. The gall bladder is adherent to the abdominal wall.

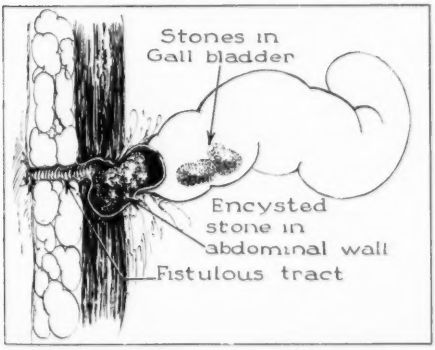

Fig. 2.—Diagram showing the position of the fistulous tract in relation to the encysted stone in the abdominal wall.

inflammatory process incident to the long-continued drainage and the associated cellulitis. However, examination of the specimen after its removal revealed the encysted gallstone lodged in the deeper layers of the abdominal wall. The stone lay in a cavity, the walls of which were quite smooth and shiny. There was only a minimal amount of bile-stained purulent material within the cavity. Bacteriological studies of this material showed the presence of nonhemolytic Escherichia coli and viridans streptococci (enterococci).

After an uneventful postoperative recovery, the patient has remained well, with no further evidence of discharge from the wound area.

COMMENT

In this instance, localized peritonitis following rupture of the gall bladder apparently caused sufficient necrosis of the abdominal wall to allow the liberated stone to work its way into the deeper musculofascial layers of the right upper quadrant. It is quite unlikely that rupture of the gall bladder would have occurred had it not been diseased. Previous bouts of inflammation probably resulted in adherence of the fundus of the gall bladder to the anterior peritoneum.

As in most cases, this external biliary fistula involved the fundus of the gall bladder. When bile is present in the discharge, such fistulas present no diagnostic problem. However, if the cystic duct is obstructed, there may be no evidence of bile in the mucus and pus draining from the fistulous tract. It is also possible, as Henry and Orr have pointed out, that the connection between the fistula and the gall bladder may become obliterated.[2]

When the etiology of the fistula is not readily apparent, detection of a gallstone in the layers of the abdominal wall or in the fistulous tract may definitively indicate its origin. However, stones liberated from a perforated gall bladder have been known to remain quiescent for long periods (up to 17 years) before becoming symptomatic.[1] Injection of an opaque medium through the fistulous opening has proved diagnostically helpful not only in detecting the presence of gallstones but also in determining the extent of involvement of other structures associated with the fistula.

SUMMARY

A case of traumatic rupture of the gall bladder with deposition of a gallstone in the abdominal wall is presented. The diagnosis was established roentgenologically by injecting an opaque medium into the fistulous tract.

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Management of Massive Melena of Undetermined Origin

CORNELIUS E. SEDGWICK, M.D.
ROBERT E. SASS, M.D.
and
LEONARD STAUDINGER, M.D., Boston

The term massive melena means the passage of large amounts of blood by rectum, frequently producing shock and a low hemoglobin and red blood cell count. This condition may necessitate several transfusions. Occasionally a patient who has had repeated gastrointestinal studies and exploratory celiotomies which have failed to reveal the source of bleeding comes for treatment of massive melena. This problem has been discussed by others, particular Stone,[1] of Baltimore, and such a case has been presented previously from this clinic by Norcross and Cattell.[2] In this paper, the problem of obscure massive melena will be reviewed, diagnostic methods evaluated, and a plan of management proposed.

With such patients the immediate treatment consists of restoring the blood volume by repeated transfusions. Another attempt is then made by diagnostic methods to determine the location and nature of the lesion. The physical examination will aid in ruling out any systemic diseases which may cause melena, such as hypertension, certain drugs, blood dyscrasias, and avitaminosis. An upper gastrointestinal x-ray series including a barium swallow will rule out the presence of esophageal varices and infectious and neoplastic lesions of the stomach and small bowel. The surgeon always keeps in mind that the bleeding may be from a strangulated hiatal hernia. This is revealed by the barium meal. An attempt is then made to evaluate the lower gastrointestinal tract by sigmoidoscopic examination and a barium enema. Such diseases of the lower bowel as ulcerative colitis and diverticulitis are eliminated. The examiner must be constantly alert to the presence of bleeding rectal or colonic polyps or true neoplastic tumors. Certain rare anomalies and tumors of the biliary and pancreatic tract which may produce massive melena must be kept in mind, but, in the main, conditions such as pancreatic or biliary calculi, neoplasms of the common bile duct or ampulla of Vater, and hemangioma of the liver are usually found only at the time of laparotomy.

At the conclusion of these studies the cause of the melena may still be undetermined. The patient continues to pass blood by rectum and requires constant multiple transfusions. Exploratory laparotomy may have been performed one or more times by a competent surgeon, the entire gastrointestinal tract carefully palpated, and no source of bleeding found. Further management in such a case presents a difficult problem. An attempt should be made to determine the level in the gastrointestinal tract at which the bleeding originates. A Miller-Abbott tube may be inserted into the upper gastrointestinal tract and slowly passed through the entire small bowel from the duodenum to the cecum; at each level samples of intestinal fluid should be obtained and tested for the presence of blood. If blood is found at a particular level of the small bowel, the surgeon can expect to find a lesion of the small bowel which can be removed and thus re-

From Lahey Clinic; Department of Surgery (Dr. Sedgwick) and Fellows in Surgery (Drs. Sass and Staudinger).

move the source of bleeding. On the other hand, if blood is not found in the entire small bowel, it must be assumed that the bleeding is from the large bowel. The problem then is to determine whether the bleeding is from the right or the left side of the colon. In a previous communication, Norcross and Cattell presented a case in which a transverse colostomy was performed so that samples could be taken from the fecal flora of both sides of the colon and the origin of the bleeding thus determined. A sigmoidoscope may be passed through the transverse colostomy both proximally and distally; evidence of bleeding may be found in this manner and the need for a right or a left colectomy determined. The case previously described from this clinic by Norcross and Cattell will be reviewed, and two additional cases in which this problem was confronted will be presented.

REPORT OF CASES

CASE 1.—An obese man, 68 years old, came to the clinic because of hemorrhage from the lower gastrointestinal tract of three weeks' duration. Four or five days after the onset of painless melena the patient sought medical advice and was admitted to another hospital. A sigmoidoscopy gave normal results except for blood in the rectum. The only positive roentgenological finding was diverticulosis of the descending and sigmoid colon. The patient was treated conservatively for seven days with multiple blood transfusions. He failed to respond and an exploratory laparotomy was performed. At operation, the upper gastrointestinal tract was normal and contained no blood. The colon was filled with old blood. Because of the diverticulosis the surgeon thought that perhaps this hemorrhage was on the left side of the colon, and a divided sigmoid colostomy was performed to rest that portion of the bowel. After operation, the patient continued to bleed from the proximal limb of the colostomy, and he was transferred to the clinic. At the time of the second laparotomy the terminal ileum, cecum, and ascending colon were resected, with an end-to-end ileotransverse colostomy. There was no evidence of further hemorrhage, and six months later the colostomy was closed. The patient has remained well.

The surgical specimen appeared microscopically to be chronic enterocolitis; definite acid-fast bacilli were not found.

CASE 2.—A 57-year-old white man was admitted to the clinic because of repeated rectal bleeding for the past two and one-half years. During this time he had been persistently anemic and had noted unsteadiness, palpitation, and easy fatigability. The patient had been hospitalized on numerous occasions and all roentgenologic and proctological examinations were negative. An exploratory laparotomy was performed, at which time no lesions were found. Later a hemorrhoidectomy was performed, without any cessation of blood loss.

Laboratory studies demonstrated severe anemia. There was no evidence of any blood dyscrasia. While in the hospital the patient continued to bleed and received numerous transfusions. An abdominal laparotomy was performed at which time no abnormality other than adhesions from previous operations was noted. Bleeding continued, and he was given transfusions of whole blood. Approximately one month after admission a Mikulicz colostomy was done on the midtransverse colon. After this procedure bleeding occurred from the proximal loop of the colostomy. Sigmoidoscopic examination was carried out from the proximal loop of the colostomy, and no definite lesion was seen; however, the ascending colon and cecum were filled with blood.

A third abdominal exploration was carried out, and blood was found in the cecum and in the last few inches of the terminal ileum. The ileum and cecum were resected, with a side-to-side anastomosis between the ileum and distal end of the colostomy. A month later the colostomy and stump of the transverse colon were closed. No abnormality was noted in the terminal ileum and cecum. Microscopic study revealed regional ileitis. No further hemorrhage has occurred.

CASE 3.—A man, 66 years old, came to the clinic because of severe anemia and repeated episodes of melena of seven years' duration. During the past three years he had been studied in numerous hospitals and repeated esophagoscopies, gastroscopies, proctoscopies, barium enemas, and gastrointestinal series gave negative results. No blood dyscrasia was found. In the year prior to the patient's admittance to the clinic two exploratory laparotomies were performed because of severe melena, but no abnormality was found. Studies at the clinic likewise were negative except for severe anemia. A Miller-Abbott tube was slowly advanced throughout the small bowel to the cecum, and serial samples of the intestinal contents were taken but were negative for occult blood. It was assumed that the hemorrhage was in the large bowel.

A third exploratory operation was performed, but no lesion was found. The terminal ileum, cecum, and ascending colon were resected on an empirical basis, and an end-to-end ileotransverse colostomy was performed. Microscopic study disclosed dilatation of the blood vessels of the submucosa in the

ileum and colon with numerous submucosal hemorrhages. Convalescence was uneventful, and there has been no trace of melena.

COMMENT

At the time of celiotomy in these three cases the surgeon was unable to find any disease of the right colon and terminal ileum that might be the source of bleeding. In view of the fact that the previous tests had indicated that, in two cases, the bleeding was coming from the right colon, right colectomy was performed. Gross examination of these specimens by the pathologist failed to reveal any changes that might explain the bleeding. However, on microscopic examination in one case typical regional ileitis was found. In the second case the blood vessels of the submucosa of the ileum and colon were dilated with numerous submucosal hemorrhages, and in the third case there was slight chronic enterocolitis consistent with cicatrizing enterocolitis. Such blind resections of the right colon and ileum without gross evidence of disease but with the knowledge that the bleeding comes from this segment of the gastrointestinal tract are somewhat analogous to subtotal gastrectomy for massive hematemesis without definite visualization at laparotomy of a bleeding ulcer but with the knowledge that the bleeding is originating from the stomach or duodenum.

SUMMARY

Occasionally, the surgeon is confronted with the problem of massive melena in which all diagnostic studies and careful celiotomy give negative results. In such cases an attempt should be made to determine the exact level in the gastrointestinal tract at which bleeding occurs by sampling intestinal contents from the stomach to the cecum. If blood is not found, it can be assumed that the bleeding originates in the large bowel. Once that is determined, a transverse colostomy may be performed to determine on which side of the large bowel the bleeding occurs. In the three cases reported, bleeding was from the right colon. Removal of the right colon and terminal ileum in these three cases has prevented further hemorrhage.

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Benign Cystic Islet-Cell Tumor of the Head of the Pancreas

Report of a Case Probably of Delta-Cell Origin

RAM A. JOSHI, M.B., Bombay, India
and
J. G. PROBSTEIN, M.D., St. Louis

In the course of a study dealing with pseudocysts of the pancreas reported by one of us (J. G. P.),[1] a case was encountered which presented clinical characteristics simulating an inflammatory cyst but which on pathological examination proved to be an unusual neoplastic lesion of the pancreas. This tumor showed cytological characteristics consistent with an islet-cell tumor, probably of delta-cell origin, as reported by Sailer and Zinninger,[2] but with the added unusual feature of cystic degeneration. This case is reported here because of its unusual pathological nature and because of certain clinical considerations which warrant discussion.

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From the Department of Pathology and the Division of Surgery, The Jewish Hospital of St. Louis, and the Department of Surgery, Washington University School of Medicine.

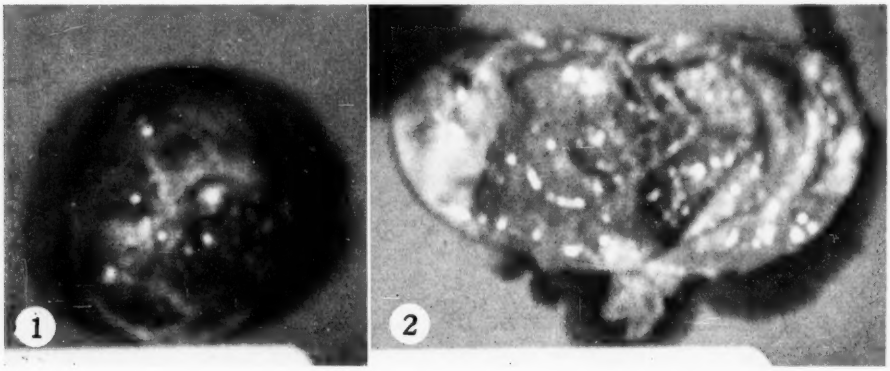
REPORT OF A CASE

A 61-year-old white woman was admitted to the Jewish Hospital in June, 1953, because of weakness, loss of appetite, loss of 25 lb. over a period of about one month, and passage of dark urine. There was no history of hematemesis, icterus, diarrhea, or bloody or tarry stools. Her past history revealed that 18 years previously she had been subjected to right radical mastectomy for carcinoma of the breast, and two years later to radical mastectomy for a malignant tumor of the left breast.

Physical examination showed a well-developed emaciated woman whose conjunctivae were slightly yellow and whose skin was questionably icteric. Heart and lungs were within normal limits. In the epigastrium and right upper quadrant a large firm mass, extending to the umbilicus, was visible and palpable. It presented a sharp lower border and a

Fig. 1.—Gross specimen, unopened. Specimen is an almost symmetrical cyst measuring about 4 cm. in diameter. Fine gray trabecular lines can be seen through the cyst covering.

Fig. 2.—Gross specimen, opened. Cyst wall measures about 3 to 4 mm. in thickness. Inner surface is trabeculated with a gray surface showing some yellow mottling.



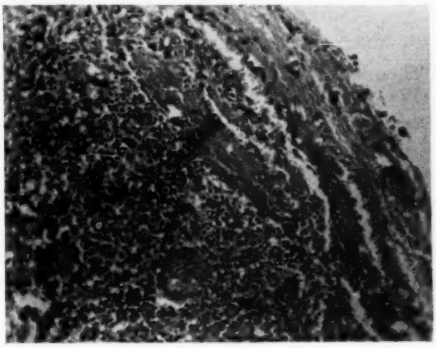


Fig. 3.—Microscopic section of cyst wall stained with hematoxylin and eosin (reduced to 7/10 of mag. approximately × 120). The inner lining of the cyst wall is composed of bands of collagenous fibrous tissue; no inner epithelial layer was found. Collections of small lymphocytes are seen within the fibrous layer. Beneath the latter are masses of cells with small hyperchromatic nuclei and occasional thick-walled small arteries.

smooth surface. The upper border of the mass coincided with the dullness of the liver, and it moved with respiration. The mass was not tender.

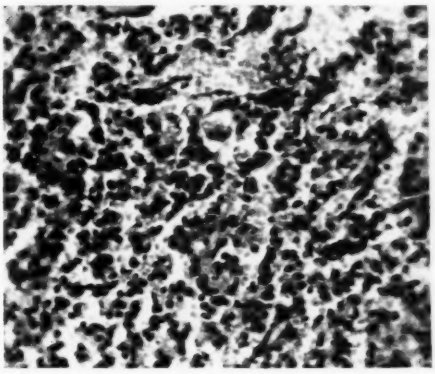
Laboratory examinations revealed hemoglobin of 9.7 gm., NPN 20 mg. per 100 cc., fasting blood sugar 89 mg. per 100 cc., blood diatase 75 Somogyi units, and icterus index 20. Stool examinations for occult blood were negative.

X-ray studies showed an elevation of the right diaphragm, which, however, appeared to move adequately. Gastrointestinal series was negative. The mass in the abdomen appeared to be continuous with the liver shadow.

An exploratory laparotomy was performed about two weeks after hospital admission, and a cystic mass 4 cm. in diameter was encountered lying in the loop of the second and third portions of the duodenum and attached only to the head of the pancreas underneath. The diastase content of the aspirated cyst fluid was only 100 Somogyi units, and report of frozen section of the liver was cirrhosis and fatty metamorphosis of severe degree (Fig. 5). The cyst was dissected out intact by blunt and sharp dissection. The external hepatic passages were not dilated. The pancreatic bed was drained by a Penrose drain through a stab wound.

The postoperative course was extremely stormy, with death on the 31st postoperative day. During this period the patient developed a pancreatic fistula with marked sloughing of the abdominal wall. She also developed marked anemia, paralytic ileus, peripheral edema, and marked electrolyte imbalance. In spite of multiple transfusions, carefully calculated intravenous fluids, and antibiotic therapy, the patient showed a markedly elevated white blood cell count, with serum proteins ranging between 3 and 4 gm. per 100 cc. and serum diastase varying between 70 and 100 Somogyi units. She finally developed severe pulmonary edema and peripheral vascular collapse and died. An autopsy was performed.

Fig. 4.—Microscopic section of cyst wall stained with hematoxylin and eosin (reduced to 7/10 of mag. approximately × 480). Epithelial cells are arranged in cords separated by capillaries. Thin bands of collagen form a supporting structure for the epithelial cords. This is the typical cellular arrangement in many areas of the cyst and corresponds to the arrangement in islets of Langerhans.



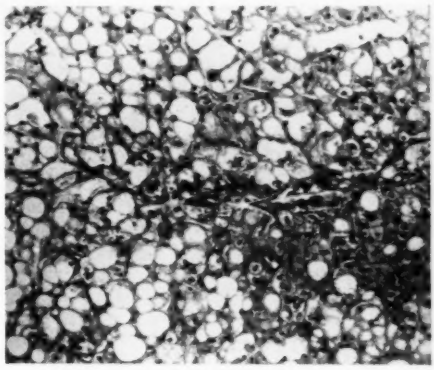


Fig. 5.—Microscopic section of liver stained with hematoxylin and eosin (reduced to 7/10 of mag. approximately × 480). Section shows a small island of liver cells with solid cytoplasm near the right lower margin. The remainder of the section shows marked replacement of cytoplasm of liver cells by fat vacuoles.

Pathologic Findings: Gross examination of the excised tumor showed a cyst measuring 4 cm. in diameter, containing a clear, straw-colored fluid. The outer wall of the cyst was whitish and smooth, but there were pink nodular areas also scattered over this outer surface (Fig 1). The inner lining showed a yellow trabeculated surface (Fig. 2).

Microscopic sections through various areas of the wall showed thin, hyaline, fibrous, ribbon-like trabeculae supporting masses of cells (Fig. 3). The latter varied moderately in size and shape and generally appeared to have the characteristics of epithelium. In places this epithelium was arranged in cords several layers in thickness, while in other areas these cells formed continuous sheets. Nuclei appeared uniformly dark blue and were generally central in location (Fig. 4). Occasional normal islets of Langerhans' were seen embedded in the capsule of the cyst. Particularly noteworthy was the almost complete absence of inflammatory cells; necrosis was present in only rare small foci.

Sections of the cyst wall were stained by a modified Gomori's stain, as applied by Nerenberg.[3] Such sections showed occasional cells in islets embedded in the capsule, which contained dust-like granules staining a typical red indicative of beta granules. Alpha cells could also be identified in such islets. However, the tumor cells of the cyst wall generally showed a clear cytoplasm devoid of granules. Occasional cells of the cyst wall contained coarser blue granules; red beta granules were never found in cells other than those in typical islets, as noted above, despite exhaustive study.

Autopsy revealed massive necrosis of the remaining pancreas and an acute toxic hepatitis superimposed on the previously noted cirrhosis and fatty degeneration of the liver. In addition there was bilateral hydrothorax and ascites of 3000 cc. of murky amber fluid. The patient also had a chronic cholecystitis and focal cerebral and subarachnoid hemorrhage. No evidence of malignant tumor was found.

In reviewing the subject of islet-cell tumors of the pancreas Anderson [4] points out that reports of the size of such tumors vary from a diameter of 1.2 mm. up to 15 cm., but the majority are between 1 and 2 cm. in diameter. The present tumor was therefore not of unusual dimensions. Degenerative changes such as fibrosis, hyalinization, and calcification, which are seen in islets of Langerhans, may also be found in islet-cell adenomas. However, we have been unable to discover any reports of cystic degeneration of benign tumors of islet-cell origin.

The cause or mechanism of this cystic degeneration is not apparent from gross or microscopic examination of this tumor. There was no noteworthy necrosis of tissue comprising the cyst wall, or was any gross evidence of acute pancreatitis or pancreatic necrosis seen at the time of surgery. The fluid within the cyst was slightly cloudy and amber, but it was not blood-tinged or did it contain fragments of necrotic tissue. However, the lack of evidence of preexisting necrosis at the time of removal of the cyst does not exclude the possibility of previous necrosis, since in our experience pseudocysts of the pancreas which have undoubtedly formed as a sequela of previous acute pancreatitis may contain fluid of similar character, and careful examination of the pancreas at the time of surgical intervention for pseudocyst may show no gross anatomic evidence of previous bouts of acute pancreatitis, despite clinical and laboratory evidence of such attacks.[1] The possibility cannot therefore be excluded that embarrassment of the blood supply to this islet-cell tumor, or possibly some traumatic incident, may have been responsible for central necrosis with liquefaction.

In general, islet-cell tumors have a morphology and arrangement resembling that of the islets of Langerhans, and individual cells are often similar to those of normal islets. By special stains both alpha and beta granules have been demonstrated in the cells of islet tumors. The arrangement of cells in anastomosing cords and bands as well as in rosettes is present in many areas of the wall of this cyst (Fig. 4) but is not as striking as in solid adenomas, probably because of distortion by the fluid content. On the other hand, the shape of the individual cells, which was cuboidal for the most part, and occasionally columnar or polyhedral, is entirely consistent with that of an islet-cell origin. The absence of specific cytoplasmic granules in this tumor, except for a few apparently normal islets incorporated in the cyst wall, would indicate an origin similar to that in a case reported by Sailer and Zinninger,[2] in which the tumor was composed almost exclusively of delta cells.

The delta cell in islets of normal mammalian pancreas was first described by Bloom.[5] The significance or function of this type of cell remains unknown. According to Gomori,[6] the delta cells are least numerous; there are seldom more than two or three per islet, and in many islets they are

completely absent. With the azan stain they show a nucleus similar to that of the alpha cell and cytoplasm containing fine sky-blue granules which sometimes stain extremely faintly or not at all. These characteristics are consistent with those seen in the cells of the present tumor. While the delta cell is generally considered a specific cell type, the possibility that it is a degenerated form of the alpha cell has not been excluded.

Both the present case and that reported by Sailer and Zinninger [2] were apparently functionally inactive. This is not an unusual clinical feature of islet-cell tumors, since, according to Anderson,[4] only about 20% of such tumors give symptoms of hypoglycemia.

The location of this cystic tumor in the head of the pancreas is clinically misleading, since the large majority of cysts of the head of the pancreas are either cystadenomas or pseudocysts. Serum diastase determinations in the present case contributed no additional information, as they were within normal limits, and they may be within normal limits in some pseudocysts and cystadenomas.[1] It is even questionable whether moderately elevated serum diastase levels would have contributed useful information, since pressure on adjacent pancreatic tissue from any cyst might give moderately elevated serum diastase levels. Despite the fact that the most frequent location of islet-cell adenomas is in the tail of the pancreas, Anderson [4] points out that the body and head of this organ may be involved. The tumor reported by Sailer and Zinninger was malignant and was located in the body and tail of the pancreas, with invasion into a large vein. The patient died of metastatic tumor.[2] The present tumor, located in the head, showed none of the histological characteristics of a malignant lesion and no tumor was found on subsequent postmortem examination.

Finally, the association of cirrhosis and fatty metamorphosis of the liver may be of importance. In an as yet unreported study, Handler and Blumenthal * have noted a high percentage of associated liver pathology in persons with islet cell tumors of the pancreas.

SUMMARY

A case of cystic islet-cell adenoma of the head of the pancreas, probably of delta-cell origin, is reported. To our knowledge, this is the second report of an islet tumor of this cell type and apparently the first report of cystic degeneration in a benign islet-cell adenoma. The previous case of a delta-cell tumor, reported by Sailer and Zinninger, showed invasion of a large vein and the patient died of metastatic tumor.

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Intravenous Urography

Improved Technique Using Dimenhydrinate (Dramamine) to Prevent Undesirable Side-Effects; Preliminary Report

DAVID ROBINSON, M.D.
and
JEROME M. VAETH, M.D., Savannah, Ga.

Intravenous urography is becoming an increasingly safer procedure as improvement in the iodine-containing contrast media has been developed.[1] With decreases in serious reactions to patients, there has been a corresponding decrease in mild but unpleasant side-effects, such as nausea, vomiting, vertigo, flushing, urticaria, and arm pain. These untoward reactions, while by no means serious, cause the patient concern and discomfort and often indirectly influence the quality of the roentgenograms.

Recently, investigators have reported that antihistaminic preparations administered before I.V. pyelography have decreased the incidence of side-effects. Bryne and Melick,[2] administering oral diphenhydramine (Benadryl) before the injection of a 70% solution of acetrizoate (Urokon) decreased undesirable side-effects of urticaria and excessive lachrymation completely, but the incidence of nausea or vomiting did not diminish. Bohne and Christeson, using antihistaminic premedication, decreased the incidence of all side-effects to 4%, in contrast to the 17% rate of reactions when antihistaminics were not used.[3]

In the series of Friedman, Lobsenz, and Jauernig, the rapid injection of iodomethamate (Neo-Iopax) diminished complaints of arm pain markedly, but the incidence of nausea or vomiting was 11.5% in 200 cases.[4]

A study by Armbruster and Helfke correlated the relationship of rate of intravenous injection of dye (in their study, iodomethamate) with the frequency and type of reaction.[5] An inverse relationship was found to exist between the side-effect of arm pain and the rate of injection; nausea and vomiting were directly related to the rate of injection. The more rapid the injection rate, the lower the incidence of arm pain above the injection site but the greater the frequency of occurrence of nausea and vomiting.

According to Richardson and Rose, nausea and vomiting is caused by the central effect of the iodine contrast media (in their studies, Urokon) on the vomiting mechanism.[6]

Dimenhydrinate, β-dimethylaminoethyl benzohydryl ether 8-chlorotheophyllinate (Searle), has as its action the prevention of nausea, vomiting, and vertigo and acts centrally.

In our experience with the various contrast media, we feel that the most frequent reactions we see are central in type, i. e., nausea, vomiting, and flushing. In this preliminary study we are presenting a series of 51 unselected cases in which dimenhydrinate was administered intravenously prior to the injection of the contrast medium. Our control group consisted of 51 unselected cases receiving similar contrast media but no premedication.

TECHNIQUE

All patients received the usual preparation for intravenous urography, i. e., catharsis and dehydration. Each patient was questioned regarding previous intravenous pyelography, and, if it had been

Consultant, U. S. P. H. S. (Dr. Robinson); Resident in Radiology, Penrose Cancer Hospital, and military leave of absence to U. S. P. H. S. (Dr. Vaeth).

given, the side-effects were noted. Blood pressure, pulse rate, and respiratory rate were recorded.

In the control group of 51 patients, 30 cc. of iodomethamate was injected rapidly, as recommended by Friedman and co-workers.[4] The patients' blood pressure, pulse, and respiration were again recorded at the completion of the injection. The patient was both observed and questioned for untoward effects.

In the dimenhydrinate group, each of 51 patients was injected with 10 cc. of a 1:10 isotonic solution of dimenhydrinate over a two-minute period. Within the following five minutes, 30 cc. of iodomethamate was administered within 30 to 60 seconds, as recommended by Friedman, Lobsenz, and Jauernig.[4] No sensitivity tests were performed. We are in agreement with other workers who feel that the value of sensitivity testing is largely limited to its possible medicolegal significance.

After the injection of dye, the patient was again carefully questioned and observed for possible visible side-effects and blood pressure and pulse and respiratory rates were recorded.

RESULTS

In our control series the reactions elicited corresponded closely to those reported by Armbruster and others[5] and Bohne and Christeson.[3] Approximately 20% suffered nausea, vomiting, and flushing. Only two patients had arm pain noted in the axilla above the injection site, this lasting but two to four minutes.

In the 51 patients premedicated with dimenhydrinate there were no incidents of nausea, vomiting, or flushing. There were three incidents of transitory axilla pain. Two of this group were patients from our control group compiled earlier and had previously suffered marked nausea, with vomiting. There were no significant differences in blood pressure, pulse, and respiratory rates recorded in the control and the premedicated groups.

Coincident with the complete absence of nausea, vomiting, flushing, vertigo, or urticaria in the dimenhydrinate group was noted an appreciable improvement in the quality of the roentgenogram—notably for the decrease in bowel gas, although both groups had similar catharsis and dehydration preparation before intravenous pyelography. We feel that this improvement was due to decreased air swallowing in the premedicated group, air swallowing occurring commonly in patients suffering marked nausea and vomiting.

CONCLUSIONS AND SUMMARY

While acknowledging the relatively few uses submitted in this preliminary study, we feel that the decreases in side-effects of nausea, vomiting, flushing, and arm pain and improvement in quality of roentgenogram warrant further investigative study on a larger scale with more control cases and placebo control cases and the use of several of the more popular contrast media. One of us (J. M. V.) is at present conducting just such a survey comparing the effects of dimenhydrinate and placebo injections with the use of acetrizoate and iodomethamate contrast media.

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Operation for Duodenal Ulcer After Inadequate Surgery

MAJOR JACOB F. SCHIRMER (MC), U. S. Army
and
COLONEL WARNER F. BOWERS (MC), U. S. Army

Experimental and clinical observations over a period of many years have brought about general agreement on the basic principles involved in surgical operations performed for the cure of duodenal ulcer. Much has been written concerning the importance of adequate initial surgical treatment of the disease. Still, patients are seen again and again, in the large centers, who were operated upon initially in an inadequate manner. At Brooke Army Hospital, 117 patients have been operated upon for duodenal ulcer disease in the last two years, 14 of these patients having been operated upon previously for this disease, and most of the initial operations having been performed at other institutions. In most of these instances the recurrence or the continuation of the disease was due to the choice of a poor surgical procedure at the initial operation or the improper application of a satisfactory operation. A few of the patients required additional surgery despite apparently adequate initial operations. We feel, however, that this incidence of recurrent ulcer disease warrants an attempt at improvement. One need but consider the complications which result from inadequate initial duodenal ulcer surgery and the difficulties inherent in the subsequent management of these patients to realize the dire necessity to treat these patients adequately when first they are considered to be surgical candidates.

In this paper we shall attempt to restate the requirements and to discuss the rationale for a satisfactory operation for duodenal ulcer, to show a manner in which these requirements may be fulfilled, to illustrate, by means of case reports, some of the complications of inadequate initial surgery for this disease, and to describe the manner in which these patients were treated.

AN ADEQUATE OPERATION FOR DUODENAL ULCER

Since gastroenterostomy first was introduced by Wolfler, in 1881, and quickly adapted as a treatment for peptic ulcer,[1] improvements in surgical therapy have consisted of more and more radical attack as a result of increasing knowledge concerning the disease. At the present time, most authorities agree that subtotal gastric resection is the treatment of choice in surgical management.* Agreement as to the manner in which the gastrectomy should be performed is not as evident, however, although most writers concur in certain basic principles.

Lannin [5] states that there are two major requirements of an operation for cure of duodenal ulcer: (1) effective reduction in gastric acid, which necessitates removal of a large portion of the gastric mucosa including the antrum, and (2) establishment of the shortest possible afferent loop. Cross [6] has shown that there is a direct correlation

From the General Surgery Service, Brooke Army Hospital, Fort Sam Houston (Major Schirmer); Chief, Department of Surgery, and Chief, General Surgery Service, Brooke Army Hospital; Chief, Clinical Surgery, Medical Field Service School, and Surgical Consultant, 4th Army Headquarters, Fort Sam Houston; Professor of Surgery, Graduate School, Baylor University (Colonel Bowers).

* References 2, 3, and 4.

between the length of the afferent loop and the pH of the content; the shorter the loop the greater the alkalinity. This is thought to have a direct bearing on the tendency toward stomal ulceration. Lannin feels that the manner of duodenal stump closure is not important provided all the antral mucosa is removed.

Most writers agree that whether or not the ulcer is removed is of minor importance.† Glenn,[11] Palumbo,[12] and others feel, however, that all ulcers should be removed. There is still greater disagreement concerning the use of the antecolic versus the retrocolic anastomosis, the wide versus the narrow stoma, and the antiperistaltic versus the isoperistaltic anastomosis.

We feel that in order to assure an adequate gastrectomy, one must remove at least 75% of the stomach; or, with the finding of a greatly dilated stomach, a still larger amount, since, as shown by Finsterer and Cunha, the cardia does not dilate as readily as the remainder of the stomach. The antral and pyloric mucosa must always be removed and only rarely should a mucosal resection be necessary. As was pointed out by Lewisohn,[13] Finsterer recommended this procedure only "if the ulcer is of such size that it reaches from the pylorus to the papilla." Ideally, the ulcer should be removed. If, however, it is thought to be inadvisable, owing to marked fibrous adhesions, acute inflammation, or position, this dissection is not attempted. We find it possible to remove the ulcer in about 90% of the cases. We perform "no loop" or short loop retrocolic gastrojejunostomies routinely in order to make the afferent loop as short as possible. In the presence of a congenitally short transverse mesocolon, marked inflammation in this area, or other contraindication, an anterior gastrojejunostomy is used. We do feel, however, that this results in a considerably longer afferent loop, despite statements to the contrary by advocates of the anterior anastomosis. A stoma approximately 2.5 cm. in diameter in an antiperistaltic anastomosis is used. The small stoma apparently reduces the incidence of the dumping syndrome.‡ The antiperistaltic anastomosis is used simply because a shorter, straighter afferent loop is obtained in this manner. Actually, since the gastroenterostomy is an end-to-side anastomosis, the term "iso-" and "anti-peristaltic" are probably not descriptive terms and really have no meaning.

The type of subtotal gastrectomy described is a logical and rational procedure which has proved its value. Despite the tomes of literature which have been written decrying inadequate, less radical procedures,[16] patients will continue to appear for treatment after having been treated in an inadequate manner. These patients present peculiarly different problems in management. The following case histories will serve to illustrate some of these problems and the manner in which they were handled at Brooke Army Hospital.

CASE REPORTS AND COMMENTS

The following 14 cases are grouped according to the operative procedure which we found necessary in order properly to correct the condition found at operation. All of these patients were seen at Brooke Army Hospital since September 1952, and most had been operated upon elsewhere for duodenal ulcer.

In the first group of five patients we were able to accomplish an 80% gastrectomy, reestablishing gastrointestinal continuity by means of a short or "no loop," posterior gastrojejunostomy with a small or Hofmeister type stoma (Tables 1 and 2).

CASE 1.—This 45-year-old white woman was admitted to Brooke Army Hospital Sept. 4, 1952, with an 11-year-old history of recurrent bouts of gastrointestinal hemorrhage and x-ray evidence of duodenal ulcer for the preceding two years. On June 23, 1952, the patient was operated upon at another hospital, where a duodenal ulcer was found, but since the operator felt that it was inactive, no corrective procedure was performed. Postoperatively the patient continued to bleed and was admitted to this hospital with a hemoglobin of 8 gm. On Oct. 29, 1952, operation again revealed a duodenal ulcer, which was treated by an 80% gastrec-

† References 7, 8, 9, and 10.

‡ References 14 and 15.

TABLE 1.—*Summary of Fourteen Cases of Duodenal Ulcer Requiring a Second Operation after an Inadequate Initial Procedure*

Case No.	Previous Operation and Result	Second Operation and Result	Indication for First Operation	Indication for Second Operation	Time from First to Second Operation	Date of Second Operation
1	Exploration only; no improvement	Partial gastrectomy; asymptomatic *	Hemorrhage	Hemorrhage	4 mo.	10/29/52
2	Pyloroplasty; partial relief	Relief of symptoms; dumping syndrome *	Abdominal pain	Intractability; hemorrhage	1 yr.	11/24/52
3	Pyloroplasty; no improvement	Asymptomatic *	Intractability; massive hemorrhage	Intractability; massive hemorrhage	8 yr.	2/16/53
4	Vagotomy with excision of ulcer; no relief	Asymptomatic *	Intractability	Intractability	2½ yr.	8/31/53
5	Retrocolic gastrojejunostomy; relief 8½ yr.	80% gastrectomy with gastrojejunostomy at site of excision of jejunum from old anastomosis; slight dumping	Intractability	Intractability; stomal ulcer	9 yr.	2/15/54
6	Vagotomy and antecolic gastroenterostomy; relief 1 yr.	With end-to-end jejunojejunostomy distal to gastroenterostomy; asymptomatic *	Intractability; massive hemorrhage	Intractability; massive hemorrhage; stomal ulcer	2 yr.	12/23/52
7	Pyloroplasty followed by gastroenterostomy; no relief	With end-to-end jejunojejunostomy distal to gastrojejunostomy; asymptomatic *	Intractability	Obstruction	3 mo.	7/20/53
8	Inadequate gastrectomy with antecolic gastrojejunostomy and jejunojejunostomy of afferent and efferent limbs; no relief	With end-to-end jejunojejunostomy distal to gastrojejunostomy and splenectomy; asymptomatic *	Hemorrhage	Intractability; hemorrhage; stomal ulcer	4 yr.	10/30/53
9	Gastroenterostomy; no relief	80% gastrectomy with Roux-en-Y gastrojejunostomy; occasional epigastric distress and weight gain	Intractability; hemorrhage	Massive hemorrhage	30 yr.	12/ 9/53
10	Vagotomy followed by gastroenterostomy (retrocolic); no relief	80% gastrectomy with Roux-en-Y gastrojejunostomy; asymptomatic	Intractability	Intractability; obstruction	6 yr.	1/22/54
11	80% gastrectomy with antecolic narrow stoma; relief of hemorrhage; continuance of distress	Vagotomy; asymptomatic	Intractability; hemorrhage	Intractability	2 yr.	9/10/52
12	Inadequate gastrectomy with antecolic isoperistaltic anastomosis; severe dumping; repeated hemorrhage	Stomal ulcer oversewn; followed later by vagotomy; moderate dumping	Intractability; hemorrhage	Massive hemorrhage; stomal ulcer	3 wk.	5/27/53
13	50% gastrectomy with antecolic anastomosis; relief of symptoms	Resection of anterior gastric wall asymptomatic	Intractability; obstruction	Massive hemorrhage	25 yr.	6/ 1/53
14	75% gastrectomy with short loop, retrocolic, small stoma gastrojejunostomy; obstructive symptoms	Reattachment of transverse mesocolon to gastric remnant; relief of obstruction	Intractability; hemorrhage	Obstruction	4 mo.	4/ 5/54

* Partial gastrectomy with excision of 75% or more of the stomach and retrocolic, antiperistaltic "no loop" gastrojejunostomy with a narrow stoma.

TABLE 2.—*Operations Which Required Subsequent Revision*

Operative Procedure	No. of Cases	Principle Indication for Reoperation		
		Hemorrhage	Obstruction	Intractability
Pyloroplasty	3	2	1	..
Gastroenterostomy alone *	3	1	1	1
Vagotomy with ulcer excision	1	..	..	1
Gastroenterostomy and vagotomy	2	..	1	1
Gastric resection (less than 75% resection)	3	3	..	..
Gastric resection (75% or more resection)	2	..	1	1
Exploratory laparotomy only	1	1	..	..

* One case also is included under pyloroplasty.

tomy with a retrocolic, short loop, narrow-stoma gastrojejunostomy. The postoperative course was uneventful, and the patient was discharged from the hospital on the 18th postoperative day. The paitient has regained and maintained her preoperative weight and at the present time has no symptoms referable to the gastrointestinal tract.

Gastrointestinal hemorrhage, in the absence of evidence to the contrary, should be considered to be from duodenal ulcer, and it is not too radical to advocate gastric resection even in instances in which exploration fails to prove that hemorrhage is from that source. Current literature [17] indicates increasing support for this view.

CASE 2.—This 39-year-old white woman first noted ulcer symptoms in 1943, and x-ray study at that time revealed evidence of an active duodenal ulcer. In 1951 the ulcer was excised and pyloroplasty performed at another institution. Postoperatively the patient's symptoms were not alleviated. Later the same year she had her first episode of hemorrhage, which abated spontaneously but recurred several times before admission to this hospital. On Nov. 24, 1952, an 80% gastric resection was performed, and gastrointestinal continuity was reestablished by means of a retrocolic, short loop, small-stoma gastroenterostomy. The postoperative course was uneventful, and the patient was discharged from the hospital on Dec. 3, 1952. The patient has had no recurrence of her preoperative symptoms.

It has been at least 20 years since anyone seriously considered excision of the ulcer and pyloroplasty as adequate therapy for duodenal ulcer. While we continue to state and teach that we do not know the cause of ulcer, yet all medical and surgical treatment is aimed at neutralizing acid or preventing its formation. Pyloroplasty does neither of these things and ulcer excision in no way corrects the basic mechanism.

CASE 3.—This 63-year-old white man was admitted to Brooke Army Hospital Jan. 21, 1953, with a history of duodenal ulcer for more than 20 years. In 1935 the patient first had a massive gastrointestinal hemorrhage, and the same year a pyloroplasty was performed. The patient noted no relief of symptoms postoperatively and had recurrences of the hemorrhage, for which he was repeatedly hospitalized. On Feb. 16, 1953, an 80% gastrectomy was performed, with restoration of gastrointestinal continuity by means of a short loop, retrocolic, narrow-stoma gastroenterostomy. The postoperative course was uneventful.

In this case pyloroplasty was done in 1935. At that time pyloroplasty was only beginning to be questioned as a definitive procedure, and this patient who never had relief from the operation bears out the current view of its inadequacy.

CASE 4.—In January, 1951, this 59-year-old white man was operated upon elsewhere by vagotomy and excision of a duodenal ulcer because of epigastric pain intractable to medical management. Because of progression of his symptoms a 75% gastric resection with gastrointestinal continuity reestablished by means of a retrocolic, short loop, narrow-stoma gastrojejunostomy was performed on Aug. 31, 1953. The patient had an uneventful postoperative course and was discharged from the hospital on Sept. 12, 1953. There has been no recurrence of symptoms.

There seems to be no point to entering the controversy between vagotomists versus resectionists. We have always decried the tendency to do a "routine" operation for any disease process and have felt that each patient merits the treatment proper for his condition. Certainly it is simpler to learn an operation for each disease, but results in patients do not bear out the efficacy of this type of surgical practice. Here, then, is one instance in which vagotomy was unsuccessful and in which subsequent gastric resection was beneficial. We do not debate the question of incompleteness of the original vagotomy.

CASE 5.—This 59-year-old Latin American woman underwent cholecystectomy for cholelithiasis and retrocolic gastroenterostomy for duodenal ulcer at the same operation in 1945. She had a recurrence of epigastric distress three months prior to admission to Brooke Army Hospital on Jan. 25, 1954. Admission x-ray studies revealed a large stomal ulcer. On Feb. 15, 1954, abdominal exploration revealed narrowing and fibrosis of the first portion of the duodenum and a stomal ulcer in the short loop, retrocolic gastrojejunostomy. An 80% gastrectomy was performed after incision of the jejunal wall adjacent to the anastomotic site to effect division of these structures. In this manner the mesenteric side of the jejunum was left intact. This portion of jejunum was then anastomosed to the gastric remnant in a retrocolic position. Postoperatively the patient experienced mild dumping symptoms.

In this case the surgeon in 1945 was operating because of gallstones and, incidentally the duodenal ulcer was discovered.

The surgeon felt that gastric resection was a procedure of too great magnitude to add to the cholecystectomy, and therefore he compromised by doing a posterior gastroenterostomy. This probably was good judgment, but it would have been preferable to consider this as the first portion of a two-stage gastrectomy, completing the procedure when the patient's condition warranted it. Possibly, the patient refused such advice. At our operation the surgeon wished to avoid the Roux-en-Y procedure and therefore used the previous gastroenterostomy site as the location for the new stoma. This simplified and shortened the operation for a patient who was not a good risk.

In the next three cases gastroenterostomy had been done elsewhere, and at our operation it was necessary to resect the stoma, to anastomose the jejunum, and to do a gastric resection, establishing a new gastrojejunostomy.

CASE 6.—This 30-year-old white man was admitted to Brooke Army Hospital on Dec. 20, 1952. Two years prior to admission the patient underwent vagotomy and gastroenterostomy at another institution for "gastric" ulcer manifested by postprandial distress and recurrent massive gastrointestinal hemorrhage. The patient was relieved of his symptoms for almost a year postoperatively, the recurrence of symptoms being associated with wounds of the extremities incurred in combat. Medical management was apparently sufficient to alleviate his symptoms at that time, and he remained well until the day before admission, at which time he suffered a massive gastrointestinal hemorrhage which did not respond to medical management. On Dec. 23, 1952, a moderate-sized artery was found to be bleeding at the site of anastomosis. The proximal and distal loops of the gastrojejunostomy were divided and anastomosed end-to-end. An 80% gastrectomy then was performed, with a retrocolic narrow-stoma gastrojejunostomy proximal to the point of the end-to-end jejunojejunostomy. The postoperative course was uneventful.

This case again represents a failure of vagotomy over a period of time, stomal ulcer and hemorrhage being the result. Here, since anterior gastrojejunostomy for drainage had been established at the time of vagotomy, the old stoma could not be used because of our desire for a short loop. Consequently the limbs of the gastroenterostomy were excised; the jejunal continuity was reestablished by end-to-end anastomosis, and the new stoma was constructed proximally. This obviated the Roux-en-Y procedure, which we prefer to avoid whenever possible. Parenthetically it may be noted that here again the vagotomy was done by the abdominal route. This is a moot point, but we feel that vagotomy by the thoracic route is much simpler technically, allows a more complete extirpation, and is easier on the patient. Its only disadvantage is that it does not permit visualization of the site of disease.

CASE 7.—This 45-year-old white man was admitted to Brooke Army Hospital July 1, 1953, with a history of ulcer symptoms of 12 years' duration. On March 6, 1953, a duodenal diverticulectomy was performed at another installation, and on April 4, 1953, a gastrojejunostomy was performed. At the

TABLE 3.—*Results of Gastric Analysis*

Case No.	Test	Preoperative					Postoperative				
		Bile	Mucus	Total Acid	Free	Combined	Bile	Mucus	Total Acid	Free	Combined
1	Caffeine	...	..	..	..	..	0	2+	8	0	8
	Histamine	...	..	..	..	..	2+	2+	19	11	8
5	Caffeine	0	1+	28	25	3	...	...	..	..	..
7	Histamine	3+	0	29	26	3	3+	0	9	0	9
	Caffeine	3+	0	15	6	9	2+	0	8	0	8
	Insulin	3+	0	61	29	..	...	...	..	..	..
8	Histamine	2+	0	34	29	5	...	...	..	..	..
	Insulin	2+	0	32	23	9	...	...	..	..	..
9	Caffeine	...	..	..	..	..	0	0	33	31	2
10	Caffeine	1+	0	29	25	4	2+	0	12	9	3
	Average of above	...	..	33	23	10	...	...	15	8	7

time of admission to this hospital gastric retention amounted to approximately 2000 ml. daily. On July 20, 1953, at surgical exploration, the stomach was noted to be extremely large and thick-walled, and the gastroenterostomy was found to be a long loop, antecolic wide-stoma anastomosis. The limbs of the gastrojejunostomy were divided and an end-to-end anastomosis performed. An 80% gastrectomy was then performed and gastrointestinal continuity re-established by means of a short loop, retrocolic, narrow-stoma gastrojejunostomy approximately 8 in. proximal to the site of the end-to-end jejuno-jejunostomy. On Aug. 16, 1953, the patient was discharged to his own care and has had no gastro-intestinal difficulties.

As can well be imagined, this patient was loath to have this third operation for ulcer within a five-month period, but his almost complete gastric obstruction forced the issue. This case exemplifies the oft-repeated statement that a duodenal diverticulum usually is a "red herring." Here the surgeon failed to realize that duodenal ulcer was the cause of the patient's symptoms and succeeded only in producing duodenal obstruction by resection of the diverticulum. The subsequent gastroenterostomy for drainage was poorly conceived in that a long loop, anterior anastomosis was made which did not function. Here again we were able to do an adequate gastric resection in the manner advocated, avoiding the Roux-en-Y procedure as previously mentioned.

CASE 8.—In 1948, this 58-year-old white man first had massive gastrointestinal hemorrhage. Recurrences of hemorrhage occurred, and in January, 1950, a subtotal gastrectomy was performed for a benign prepyloric ulcer. After this procedure the patient was repeatedly hospitalized for recurrences of massive hemorrhage and epigastric pain. On Oct. 30, 1953, abdominal exploration revealed that only a small amount of stomach had been removed and that, in addition to the antecolic, long loop gastro-jejunostomy, there was almost immediately below it a side-to-side jejunojejunostomy. The jejunal limbs were amputated and an end-to-end anastomosis performed. A high gastric resection with splenectomy was executed, and gastrointestinal continuity re-established by means of a short loop, retrocolic, narrow-stoma gastrojejunostomy proximal to the end-to-end jejunojejunostomy. Postoperatively the patient developed a subdiaphragmatic abcess which necessitated drainage on Dec. 11, 1953. At the time of discharge from the hospital on March 19, 1954, the patient had no difficult referable to the gastro-intestinal tract and was gaining weight.

This case brings up several important points referable to the original operation as well as to our procedure. First, a "small" gastric resection was done. There is almost universal agreement that if a gastric resection is to be done at all, at least 75 to 80% of the stomach must be removed in order to control the acid factor adequately. Next, a long loop, antecolic anastomosis was made, inviting development of a stomal ulcer from the remaining acid-secreting potential plus the decreased neutralizing ability of the long jejunal loop. Finally, to make a stomal ulcer almost inevitable, a side-to-side jejuno-jejunostomy was done to shunt alkaline juices away from the stoma completely. Nothing favorable to stomal ulcer remained undone here. The case of this patient represents the only major complication in our series and indeed one of our few major complications, even if all the elective original gastric resections are included. In the process of ablating the jejunojejunostomy and establishing a short loop posterior gastrojejunostomy after gastric resection, the spleen had to be removed and the pancreas had to be dissected off of the posterior gastric wall. Pancreatic leakage was anticipated and a drain was left in the area. However, drainage was provided for inadequately, and there was accumulation of material in the region from which the spleen had been removed. A typical left subdiaphragmatic abscess had to be surgically drained and this, together with the stubborn pancreatic fistula, accounted for the lengthy hospital stay. On discharge, these conditions had been successfully controlled.

The next two cases indicate that it is not always possible to avoid the Roux-en-Y procedure, which we accept as a compromise, favoring development of stomal ulcer.

CASE 9.—In 1923, this 61-year-old white man had undergone gastroenterostomy for bleeding peptic ulcer. Bleeding and a recurrence of other symptoms followed surgery. The patient had been hospitalized repeatedly for this condition and was hospitalized from May to June, 1953, for myocardial infarction.

At the time of admission the patient was not receiving cardiac medications. Subtotal gastrectomy was planned for Nov. 27, 1953, but during the induction of anesthesia the patient sustained a cardiac arrest which was treated by massage. Within five minutes of arrest normal rhythm had been established. The chest was closed and the patient returned to the ward. Inasmuch as the operation was considered an elective procedure, it was decided that the patient would not be operated upon. On Dec. 9, 1953, twelve days after the cardiac arrest, however, the patient sustained a massive gastrointestinal hemorrhage of sufficient degree that emergency surgery was considered imperative. He was operated upon that day, no grave anesthetic difficulties being encountered. The stomach and duodenum were tremendously dilated, thick-walled, and edematous. The afferent loop of the retrocolic, long loop, isoperistaltic gastroenterostomy was found to be dilated to approximately 9 in. in diameter and contained approximately 3000 cc. of bloody gastric content. A high gastric resection was performed. Gastrointestinal continuity was reestablished by means of a Roux-en-Y procedure by end-to-end anastomosis of the stomach to the efferent jejunum, and, after resection of part of the dilated, redundant afferent loop and closure of part of the lumen, end-to-side anastomosis of a portion of the afferent jejunum to the efferent jejunum immediately below the gastrojejunostomy was done. The patient was discharged from the hospital on Jan. 6, 1954. Three months later the patient stated that he had occasional epigastric discomfort but that he weighed 10 pounds more than his preoperative weight.

Our preoperative x-ray study showing a huge density in the left upper quadrant was the cause of much speculation as to etiology, but our preoperative diagnosis was obstruction at the stoma of an isoperistaltic gastroenterostomy with dilatation of the afferent loop, which in this instance lay in the left upper quadrant. This pouch of jejunum contained 3000 cc. of material, and the tremendous dilatation extended proximally through the area of the ligament of Treitz with concomitant massive dilatation of the duodenum. Since there was no normal bowel proximally, normal jejunum distally was anastomosed end-to-end with the stomach. Then most of the circumference of the dilated pouch was excised, and this relatively normal diameter was anastomosed end-to-end in the Roux-en-Y fashion with the efferent loop. This was accepted as a compromise in an extremely poor risk patient. The fact

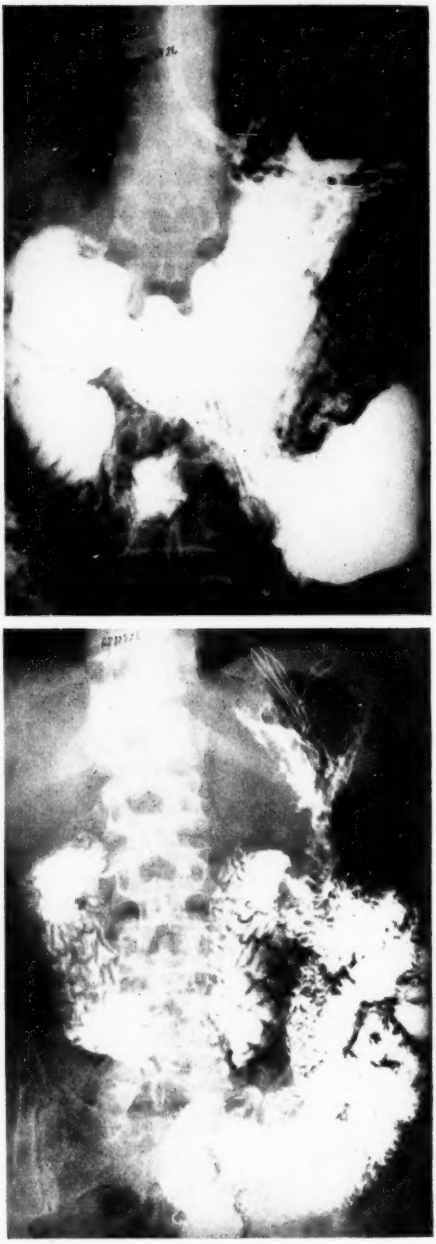

Fig. 1 (Case 9).—*A*, Preoperative x-ray, showing obstruction at the gastroenterostomy stoma with dilatation of the stomach, duodenum, and afferent limb of jejunum. The afferent limb held 3000 cc. of fluid at the time of reoperation. *B*, postoperatively there has been marked diminution of the lumen of the afferent loop.

that this patient with a precarious cardiac status and previous cardiac arrest withstood this rather formidable procedure is in itself a matter of some interest (Figs. 1 and 2).

CASE 10.—This 26-year-old white man had a history of duodenal ulcer since age 16. In 1947 a vagotomy was performed with no postoperative improvement, and the following year a gastroenterostomy was performed. Because of continuance of symptoms and increasing gastric obstruction, the patient was admitted to Brooke Army Hospital on Jan. 2, 1954. A nonfunctioning, retrocolic, isoperastalic gastrojejunostomy was found at operation on Jan. 22, 1954. After amputation of the jejunal limbs, an 85% gastrectomy was performed. An end-to-end gastrojejunostomy was performed, with the use of the efferent jejunal limb. The proximal jejunal limb which had been amputated at the ligament of Treitz was anastomosed in an end-to-side manner into the efferent jejunum just distal to the gastroenterostomy. The patient's postoperative course was uneventful, and three months later he ha ' regained his preoperative weight and was working eight hours daily as a postman. His only gastrointestinal symptoms were referable to decreased gastric capacity.

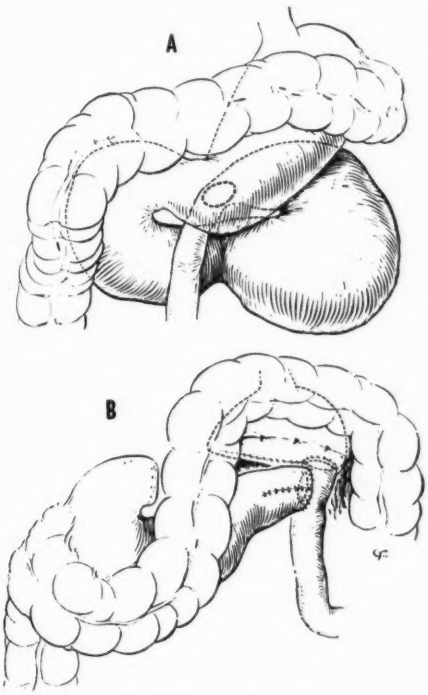

Fig. 2.—*A*, illustration of the status at the time of surgery in Case 9. A long loop, isoperistaltic gastrojejunostomy had been performed previously with a resultant torsion-obstruction at the stoma. This resulted in tremendous dilatation of the stomach, duodenum, and afferent jejunal loop.

B, Owing to this marked dilatation, it was felt unwise to perform a gastrojejunostomy proximal to the old anastomosis. Therefore, after a gastrectomy had been performed, the stomach was anastomosed end-to-end to the efferent limb of jejunum. The redundant afferent loop was then amputated close to the ligament of Treitz and the lumen reduced in size by a wedge excision of a portion of its circumference, allowing end-to-side anastomosis to the efferent loop immediately below the gastrojejunostomy. Case 10 was similar to this one.

This case again represents a failure of vagotomy complicated by a poorly constructed, nonfunctioning gastroenterostomy established at a later date in an attempt to promote drainage. It is now fairly widely conceded that if vagotomy is to be done, a drainage procedure also is indicated, but this must be adequately established to accomplish its purpose. Isoperistaltic gastroenterostomy adds the risk of torsion-obstruction at the stoma, and this is the second example of such a mechanism in our 14 cases. Here, again, the anatomical situation seemed to make the Roux-en-Y procedure mandatory.

In the next two cases, we elected to attempt to correct previous inadequate surgical treatment by adding vagotomy.

CASE 11.—This 31-year-old Negro was admitted to the hospital Aug. 27, 1952, with a history of duodenal ulcer of four years' duration. Two years prior to admission he underwent an antecolic, narrow-stoma, 80% gastrectomy. Continuing subjective symptoms without bleeding caused the patient to return for further treatment. Examination revealed gastric hypersecretion and hyperacidity; there was no evidence of stomal ulcer. Transthoracic vagotomy was performed on Sept. 10, 1952. The patient did well postoperatively and had no symptoms referable to the gastrointestinal tract.

It was our opinion that this patient with hypersecretion and hyperacidity was a good candidate for stomal ulcer and that all of his symptoms were on the basis of an incompletely controlled acid factor. Probably this represents the best indication for vagotomy in a patient who already has had gastric resection. In this instance vagotomy was a suitable adjunct.

CASE 12.—This 30-year-old Latin American man was admitted to Brooke Army Hospital on March 17, 1953, in shock due to gastrointestinal blood loss. Duodenal ulcer was known to be present since 1949. On Feb. 25, 1953, a subtotal gastrectomy was done at another institution. The patient's symptoms were partially relieved. Under medical management the hemorrhage abated, only to recur on March 24, 1953. On March 27, abdominal exploration revealed an antecolic isoperistaltic anastomosis with a very large residual gastric pouch. The duodenal stump was involved in an absorbing mass of blood and necrotic tissue. Fat necrosis was noted. Duodenotomy revealed no bleeding or ulceration. Gastrotomy revealed an actively bleeding ulcer in the gastrojejunostomy site. Owing to the patient's poor condition, the ulcer was oversewn and the operation terminated. On May 8, 1953, transthoracic vagotomy was performed. After this operation the patient did well, and x-ray studies demonstrated an adequate stoma with no evidence of a persistent stomal ulcer. On May 25, the patient was discharged from the hospital. Three months after discharge the patient had some evidence of the dumping syndrome, which could be remedied by dietary measures.

This case represents an inadequate removal of stomach tissue plus a type of anastomosis which compounds the difficulty. The patient was a poor operative risk, because each time the anesthesia was deepened sufficiently so that the operator could work there was a profound blood pressure drop not due to decreased circulating blood volume. It was not felt that re-resection was warranted, and the stomal ulcer was quickly oversewn to stop bleeding. Since this type of case again fits into the category of those for whom vagotomy is beneficial, such a procedure was carried out with good result.

The next case is an unusual one in which an unusual form of therapy was employed.

CASE 13.—This 64-year-old white man had a subtotal gastrectomy in 1928 for duodenal ulcer with obstruction. Exploratory laparotomy was performed at Brooke Army Hospital on June 1, 1953, after a two-month history of right upper quadrant pain, anorexia, fatigue, 30-lb. weight loss and massive gastrointestinal hemorrhage. Approximately 50% of the stomach had been removed at the previous operation and gastrointestinal continuity had been reestablished by means of an antecolic gastrojejunostomy. Numerous small varices of the anterior wall of the stomach were found to be oozing blood at the time of inspection. A large area of the anterior wall of the stomach including all these varices was then removed and the operation terminated. There was no evidence of portal hypertension. The postoperative course was uneventful, and on the 15th postoperative day the patient was transferred to the gastroenterology section for dietary regulation in view of the man's marked weight loss, emaciation, age, and general debilitation.

This patient, a retired enlisted man, lived alone and was not eating properly from the standpoint of quantity or quality. Pathologic examination showed a marked atrophic gastritis and the varices in the gastric wall represented relatively normal vessels made more prominent by the atrophic wall. Bleeding was by erosion. Since there was no acid factor and since there was no obstruction, it did not seem warranted to do a re-resection, nor was there good reason to change the antecolic long loop to a retrocolic short loop. Therefore a large segment of the anterior gastric pouch wall was excised. No other bleeding points or prominent vessels could be found and plastic closure of the gastric pouch was carried out. The postoperative result was satisfactory, and effort was spent largely on training the patient to eat properly.

The final patient in our series represents mechanical obstruction of the afferent and efferent jejunal loops by the hiatus in the transverse mesocolon.

CASE 14.—This 45-year-old white man was admitted to Brooke Army Hospital April 1, 1954, with the chief complaint of nausea and vomiting of three weeks' duration, a 20- to 30-lb. weight loss during that time, weakness, and muscular cramps.

On Dec. 17, 1952, a 75% gastrectomy had been performed, with a retrocolic, short loop, narrow-stoma gastrojejunostomy being used to reestablish gastrointestinal continuity. The patient was operated upon at that time for duodenal ulcer manifested by repeated episodes of gastrointestinal hemorrhage and epigastric pain. For the first few days postoperatively, the patient did well, but after considerable coughing due to a chronic bronchitis, he began complaining of rather severe abdominal cramping pain and fullness in the epigastrium. The following month the patient noted incisional herniation, which was repaired on May 8, 1953.

At the time of the last admission to the hospital, x-ray studies revealed obstruction of the afferent and efferent jejunal limbs a few centimeters from

the stoma (Fig. 3). The patient was operated upon on April 5, 1954. The transverse mesocolon was found to be obstructing both jejunal limbs approximately 7 cm. from the gastroenterostomy. The mesocolic attachments to the jejunum in this region were dissected free to a point well up on the wall of the gastric remnant. The transverse mesocolon was then attached in this region.

This case represents the only patient in this series and in all cases treated at Brooke Army Hospital in the last five years to require a second operation after the procedure

Fig. 3 (Case 14).—Preoperative x-ray showing obstruction of both afferent and efferent jejunal loops by the transverse mesocolon.

we have recommended in this paper. This fact, it would seem to us, indicates that the utilization of the retrocolic, short afferent loop, narrow-stoma anastomosis in conjunction with at least a 75% gastrectomy results in the performance of an adequate operation for duodenal ulcer.

SUMMARY

The requirements for an operation to cure duodenal ulcer are reviewed, and a logical procedure to fulfill these requirements is suggested. The operation consists of a gastric resection, amounting to at least 75% of the organ, and a short loop, retrocolic, narrow-stoma gastrojejunostomy.

Fourteen case histories are presented. These cases represent patients who had duodenal ulcer and were operated upon for their disease initially in an inadequate manner.

The surgical management of these cases at Brooke Army Hospital is discussed. Insofar as possible, the existing gastrointestinal mechanism resulting from the previous operative attempt was revised in such a manner as to provide an adequate gastrectomy with a short afferent loop, retrocolic, narrow-stoma gastroenterostomy.

The importance of adequate initial surgical treatment of duodenal ulcer is emphasized, and the manner in which inadequacies are handled at Brooke Army Hospital is described.

CONCLUSIONS

1. Inadequate initial surgery for duodenal ulcer must be condemned.

(*a*). Adequate surgical therapy requires that all the antral mucosa and a large portion (at least 75%) of the body of the stomach be removed.

(*b*). The afferent jejunal loop should be made as short as possible.

(*c*). Retrocolic gastrojejunostomy can be performed with a shorter afferent loop than can an antecolic gastrojejunostomy.

(*d*). To reduce the incidence of the dumping syndrome, a narrow gastrojejunostomy stoma should be constructed.

2. Surgery for cure of ulcer disease following previous inadequate attempts must be individualized.

(*a*). Conversion of an inadequate operation into an adequate one is best done by revision of the operation to conform to the criteria listed above.

(*b*). Roux-en-Y procedures should be avoided if possible but may be of value in isolated instances.

(*c*). Vagotomy is of value in the treatment of stomal ulcer if an adequate gastrectomy

was performed previously or if revision of the gastrectomy is contraindicated.

3. Cardiac arrest at the time of one surgical procedure is not necessarily an absolute contraindication to necessary surgery at a later date.

In the interests of brevity, many controversial points are covered herein by a seemingly dogmatic statement without elaboration or explanation. Elucidation of these additional features, such as the dumping syndrome, will be attempted in a subsequent series of publications.

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Foreign Bodies in the Common Duct

JOHN J. BYRNE, M.D.
and
CHARLES J. MEKELATOS, M.D., Boston

Foreign bodies in the common duct are such rare surgical entities that a recent case observed at the Boston City Hospital is presented.

REPORT OF A CASE

M. R., (B. C. H. No. 1502986) a 67-year-old white man admitted to the hospital because of jaundice.

This patient, troubled with indigestion for six months, noted a dull persistent epigastric pain approximately five days before entry which radiated to the left lower quadrant and was associated with nausea and scleral jaundice. Three days before entry dark-colored urine was noted, but no color changes were observed in the stools.

Physical examination was essentially negative except for slight tenderness in the right upper quadrant of the abdomen associated with jaundice.

Laboratory findings revealed bile in the urine, gray stools, icteric index 48, urine urobilinogen 0, prothrombin time normal, cephalin-flocculation 3+, formol-gel negative. A duodenal aspiration revealed an absence of carcinoma cells.

A flat plate of the abdomen was normal, and two Graham-Cole series showed no filling of the gall bladder. A gastrointestinal series demonstrated a flattening of the duodenal cap which may have represented an extrinisic pressure defect from an enlarged gall bladder.

Operation for cholelithiasis and choledocholithiasis was performed, and numerous gallstones were found in the gall bladder. After cholecystectomy the common duct was explored, and several soft gallstones were found in the center of which was embedded a 3-cm. foreign body (Figure). After the common duct was irrigated with saline solution a

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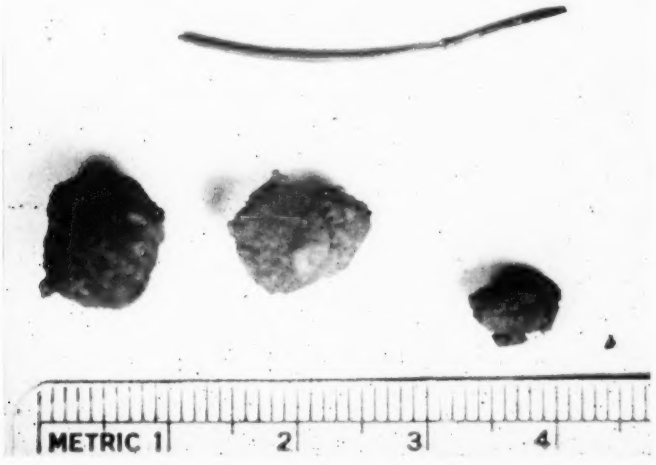
From the Third (Boston University) Surgical Service, Boston City Hospital.

Associate Professor of Surgery (Dr. Byrne) and Assistant in Surgery (Dr. Mekelatos), Boston University School of Medicine.

Woody fiber discovered in common duct with common duct stones which had formed around the fiber.



T-tube was placed in the common duct, a drain placed in Morison's pouch and the wound closed in the usual fashion, with the tubes brought through a stab wound. The patient had a relatively normal postoperative course, and a cholangiogram on the 14th postoperative day demonstrated no obstruction of the biliary tree, with prompt passage of the dye into the duodenum. The T-tube was removed and the patient discharged without incident on the 18th postoperative day.

The foreign body grossly resembled a woody fiber. It was mounted in glycerin and examined with a polarizing microscope and found to contain no cholesterol crystals or free cholesterol esters. Microscopic studies were made of the foreign body, which revealed a fibrillar nature with no definite cellular structure observed.

COMMENT

Parasitic Foreign Bodies.—Ascariasis is one of the commonest worm infestations in man. Occasionally this worm may invade the biliary tract producing obstruction with the usual sequelae of infection, biliary cirrhosis, and pancreatitis.

Estrada and Garcia [1] reported in 1942 what was probably the first case of removal of an adult worm from the common bile duct followed by recovery of the patient. Yang and Laube [2] reported 24 cases of this disease and reviewed the characteristic clinical findings. There was usually epigastric or right upper quadrant pain, and at times this had a characteristic distending quality, in contrast to the stabbing knife-like pain of cholelithiasis. One of their patients stated that he thought something was crawling around inside him. Five of the 24 cases were jaundiced. Physical findings revealed epigastric and right upper quadrant tenderness and spasm. The stools were usually positive for Ascaris ova. Practically all the cases underwent surgery with a preoperative diagnosis of cholecystitis and cholelithiasis, although a correct preoperative diagnosis was made in four. At operation about one-third of a 20-cm. worm usually projected into one of the hepatic ducts, while the remaining two-thirds extended down the common duct into the duodenum. In several of the cases it also extended into the gall bladder. There were 3 deaths in the 24 cases: one from Friedländer bacillus septicemia, one from multiple liver abscesses, and one from bilateral pneumonia.

Altounyan [3] reported an interesting case of this disorder, where 21 large ascarides were removed from the common duct and subsequently three more evacuated from the common duct tube.

Fasciola hepatica, the well known liver fluke, usually affects sheep, producing a perihepatitis with cholangitis, liver abscess, and intralobular cirrhosis. Man becomes infected when eating infested water cress. These parasites excyst in the duodenum and migrate up the common duct into the biliary passages, where they grow to maturity. They may produce a chronic cholangitis with a mild degree of cirrhosis. There is occasionally a febrile state with hepatitis, perihepatitis, and pain in the region of the liver.

Probably the first case where a liver fluke was removed from a human being was reported by Manson-Bahr and Walton.[4] This patient was a 54-year-old woman who had the habit of chewing grass and eating water cress. She had a previous cholecystectomy and developed recurrent pain in the epigastrium radiating to the back. Exploration revealed the common duct to be considerably dilated, and when it was opened a flattened folded and relatively soft object was removed, which proved to be a fully adult Fasciola hepatica containing eggs and measuring 23 × 13 mm.

Murphy and Pascall [5] reported three similar cases. Ramage [6] reported another case and gave figures from the abattoir in Leek, England, where it was found that 26% of the livers of sheep and cows were infected by this parasite.

Nonparasitic Foreign Bodies.—The commonest cause of nonparasitic foreign body in the common duct is an object left behind after surgery. The commonest object reported has been a portion of a catheter or a T-tube inserted for common duct drainage.*

* Fedoroff, cited by Thorek [10]; references 7 to 10.

In two of these cases the tube fragment remained for 25 years before symptoms necessitated removal.† Gauze sponges or swabs have occasionally been overlooked in the abdomen and migrated into the common duct.‡ Very rarely an instrument has broken off and was left in the common duct as reported by Crawford and Stewart.[12] In this case the blade of a hemostat measuring 4 cm. in length was found in the common duct producing a fistula between the common duct and the duodenum 10 years after cholecystectomy. An x-ray on this patient demonstrated the metallic foreign body present in the right upper quadrant prior to surgery.

The second pathway for a common duct foreign body is penetrating trauma. Eastman [13] reported a short sewing needle in the gall bladder forming the nucleus for gallstones. He reasoned that this needle penetrated the pylorus or duodenum and perforated into the gall bladder. Goldhan § recovered a copper bullet in the cystic duct of a 30-year-old man who had sustained a bullet wound 14 years prior to surgery. Hurt [14] reported a 19-year-old soldier who was wounded by a mortar shell fragment six months' prior to surgery. Because of a persistent biliary fistula with development of clay-colored stools, a laparotomy was performed and a ½ by ¼ by ¼ in. fragment of metal removed from the common duct. Hebert and Swarts [15] reported a somewhat similar case of a 29-year-old man who entered the hospital because of obstructive jaundice. The man had been wounded in action nine years prior to this admission, and it was noted that a machine gun bullet was left in the right lobe of the liver. A scout film of the abdomen just prior to operation for relief of the obstructive jaundice demonstrated a 3-cm. shadow just below the right leaf of the diaphragm whose shape was consistent with that of a bullet. On exploration of the common duct a 3.5 by 1.5-cm. machine gun bullet was removed from the right hepatic duct.

The third means whereby a foreign body can gain entrance to the common duct is by way of the ampulla of Vater. Lemierre and Pollet || describe an incredible case of an insane woman who swallowed a spoon handle 13.5 cm. in length. A laparotomy performed because of a possible liver abscess revealed a completely oxidized spoon handle in the common duct extending into the liver. Cooke [16] discovered a piece of wire in the common duct. Baumgartner and Lasky [17] removed a large common duct stone embedded in which was a cherry stem. The woody fiber reported in the present article no doubt gained access to the common duct through the ampulla of Vater.

† References 9 and 10.

‡ Opel, cited by Thorek [10]; reference 11.

§ Cited by Thorek.[10]

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SUMMARY

Foreign bodies are rarely found in the common duct. A case is reported wherein a woody fiber was discovered in the common duct around which numerous common duct stones developed.

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The Surgical Management of Postanastomotic and Postgastrectomy Malfunctions

M. E. STEINBERG, M.D., Portland, Ore.

Patients with a variety of minor or major postgastrectomy disturbances are most frequently designated as mild or severe dumpers. Theories regarding causation of such disturbances are most frequently based on a number and variety of manifestations ascribed to the precipitate evacuation of unprepared foodstuffs into the efferent jejunal loop (dumping). A searching review of the case histories of 33 patients personally subjected to corrective operations because of crippling postanastomotic and postgastrectomy side-effects with the analysis of results after surgery affords additional support to my previous contention that malfunctions other than those caused by dumping are rather frequent.* A number of such malfunctions can be either prevented or eventually rectified by definitive surgical techniques.

Issues involved in the causation of postanastomotic and postgastrectomy side-effects may be attributed to one of three separate mechanisms of malfunction: (1) Gastric pouch malfunction; (2) Dumping malfunction (efferent loop syndrome, Wells and Welbourn [16]); (3) Reflux malfunction and afferent or efferent enteric loop stasis.

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From the Department of Physiology, University of Oregon Medical School and the Emanuel Hospital.

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* References 10, 11, and 12.

GASTRIC POUCH MALFUNCTION

The gastric pouch has to a greater or less degree lost its retentive and churning functions, serving largely as a connecting link or funnel between the esophagus and the jejunum, particularly when the pouch is minimal, when the stoma is wide, and when only fluids are swallowed. Food escapes into the jejunum in large quantities, with scant chance of being predigested or its temperature and osmotic pressures properly regulated. Evacuation from the gastric pouch is brought about mainly by gravity, that is, by change in the patient's position or change in the abdominal pressures. Other factors which influence evacuation from the gastric pouch depend on the tonus of its gastric walls, the remaining innervation, and on the degree of hydrostatic pressure. The width of the anastomosis and the consistency of the gastric contents influence the rapidity with which the gastric pouch empties itself. Liquid contents leave the gastric pouch rapidly, even in the presence of a very small stoma.

An indefinite though considerable number of patients are unable to partake of a heavy meal because of a feeling of fullness, nausea, or vomiting. Several patients have stated that an extra mouthful of food after a feeling of satiety would bring on profuse perspiration, nausea, and palpitation.

DUMPING MALFUNCTION (EFFERENT LOOP SYNDROME)

When the symptoms are considered to originate in the efferent jejunal loop, Wells and Welbourn [16] designate the condition as the efferent loop syndrome.

Because the gastric pouch lacks propulsive peristaltic action, it becomes obvious that the gastroenteric stoma should always be placed in the most dependent part of the gastric pouch. A measure of dumping must be accepted as unavoidable. Aggravation of postprandial symptoms would inevitably result from a very small stoma or one placed at the lesser curvature of the gastric pouch. An exceptionally wide stoma, on the other hand, is also not advisable because it is likely to cause precipitous evacuation of solid gastric contents and because of the probability of causing reflux of intestinal contents into the gastric pouch.

Disabling symptoms from dumping are frequently observed in underweight viscer-optotic patients, in those with a history of bowel unrest, in the emotionally hypersensitive and psychoneurotic, and in patients in whom the operation was not indicated.

The mechanisms involved in certain postcibal symptoms as well as a deficiency in the utilization and absorption of certain foods, the inability to gain weight, intolerance of certain foods, diarrhea, steatorrhea, and hypoglycemia are related to the loss of gastric functions and to the rapid entrance and transit of abnormally large quantities of foods, inadequately prepared, into the efferent loops of the small bowel. Adlersberg and Hammerschlag [1] introduced the term "early postprandial symptoms" as distinguished from "late postprandial symptoms."

Early Postprandial Symptoms.—Early postprandial symptoms vary in intensity. Soon after a meal the patient may complain of fullness, nausea, eructation, and flushing of the face. The feeling of drowsiness and lassitude and a sense of warmth, together with beads of perspiration, may similate an attack of syncope. At first the patient may be greatly alarmed, but he soon learns that if he assumes a recumbent position the symptoms disappear rather suddenly after a shorter or longer period. It is claimed that the relief given by recumbency is due to the slowing of evacuation from the gastric pouch. It may also be assumed that symptoms caused by disturbances in vasomotor equilibrium with cerebral ischemia because of a splanchnic overflow of blood are ameliorated after the patient assumes a recumbent position. The above symptoms together with intolerance of some foods, such as milk, concentrated sugars, and carbohydrates, have not been satisfactorily explained. It is believed that intolerance of certain foods may also indicate an allergic phenomenon due to absorption of incompletely broken-down proteins.[2] Distention of the jejunum by outpouring of excessive jejunal secretion, caused by the presence of hypertonic foodstuffs, has been advanced as the cause of some of the symptoms due to dumping.[6] Others have considered the early postprandial symptoms from dumping as a splanchnic or vasomotor disturbance, initiated by some sort of a neurovisceral reflex. According to Wells and McPhee,[15] increased tension in the jejunum with hyperperistalsis rather than distention is the mechanism which causes disturbances from dumping.

Diarrhea and steatorrhea are believed to be caused by jejunitis, altered bacterial flora, or lack of fat absorption because of the increase of intestinal motility and failure of the food to be thoroughly mixed with bile and pancreatic juices. It will become apparent in the forthcoming pages that pale stools, diarrhea, and steatorrhea are also symptomatic of reflux and enteric loop stasis malfunctions.

A fall in serum potassium during dumping attacks has recently been demonstrated by Smith.[8]

Late Postprandial Symptoms.—The late postprandial symptoms because of dumping take place between meals when the gastric pouch is empty. The patient complains of weakness, palpitation, trembling, shortness of breath, and a flickering before the eyes. Such symptoms may appear with an alarming swiftness and may end in syncope. Butler [3] submits with caution that his studies indicate that the cause of the syndrome may be temporary inhibition of glycogenolysis in the liver in response to a high degree of portal hyperglycemia due to rapid absorption of carbohydrates.

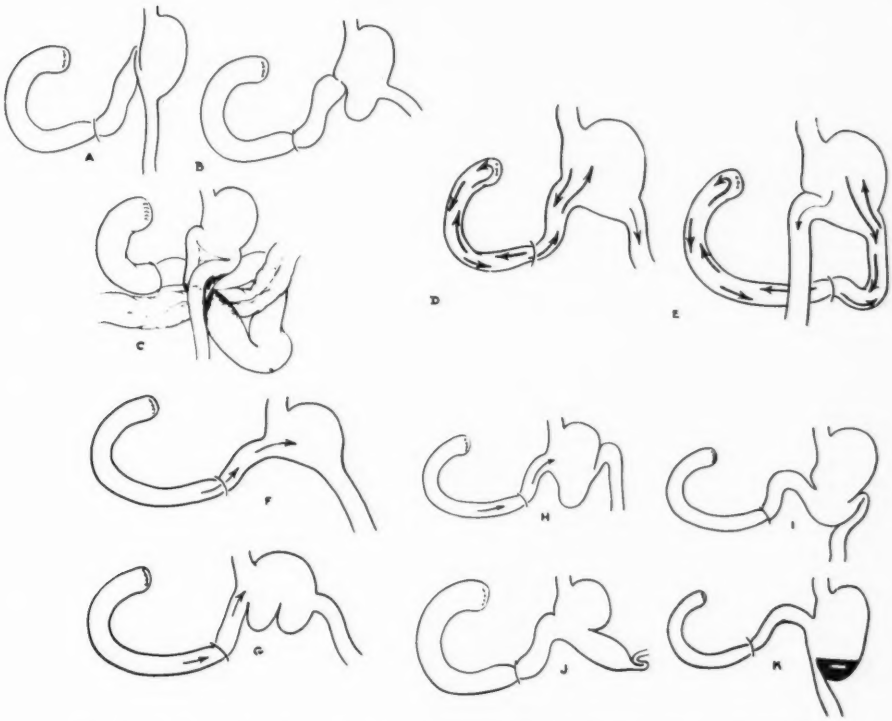


Fig. 1.—*A*, *B* and *C*, afferent loop stasis with reflux: *A*, kinking of the afferent jejunal loop on the lesser curvature; *B*, partial stenosis at the afferent angle of the stoma; *C*, antecolic anastomosis with compression of the afferent jejunal loop by the jejunal mesentery. Partial obstruction may also result from the compression of the jejunal loop riding on a distended transverse colon.

D and *E*, pedulum reflux: *D*, pendulum reflux in a Reichle-Polya anastomosis with the proximal jejunal loop from right to left; *E*, pendulum reflux is more commonly observed in an antecolic gastrectomy with the jejunal loop directed from left to right.

F and *G*, uncomplicated isoperistaltic reflux: *F*, Reichle-Polya anastomosis with a wide stoma; *G*, same as *F* but with redundancy and sacculation of the jejunal wall opposite the stoma.

H, *I*, *J*, and *K*, reflux complicated by efferent loop stasis: *H*, high attachment of the distal jejunum on the greater curvature with the jejunal sacculation opposite the stoma; *I*, kinking of the distal jejunal loop from adhesions on the loop to the jejunal segment opposite the stoma; *J*, kinking of a distal jejunal loop some distance from the stoma; *K*, reflux with "cup-and-spill" mechanism of malfunction. This is most frequently seen in gastrectomies with large gastric pouches or where the stoma is placed at the lesser curvature of the gastric pouch.

REFLUX MALFUNCTION AND AFFERENT OR EFFERENT ENTERIC LOOP STASIS

Reflux malfunction and afferent or efferent loop stasis may be considered in the following categories: (*A*) Afferent loop stasis with reflux (Steinberg, 1940;[10] Wells and Welbourn [16]) (Fig. 1*A*, *B*, and *C*); (*B*) Afferent loop stasis without reflux (Steinberg;[10] Wells and McPhee [15]); (*C*) Pendulum reflux (Steinberg) (Fig. 1*D*, and *E*); (*D*) uncomplicated isoperistaltic afferent loop reflux (Steinberg) (Fig. 1*F* and *G*); (*E*) reflux with malfunction at the efferent angle of the stoma or at the efferent jejunal loop (Fig. 1*H*, *I*, and *J*); (*F*) cup-and-spill mechanism of malfunction (Fig. 1*K*), and (*G*) combination of mechanisms of reflux malfunction.

I first called attention in 1940 to the deleterious effects from reflux of bile and pancreatic juice into the gastric pouch.

The presence of bile and pancreatic juice which may stagnate in the stomach may be a positive factor in gastritis and some of the dyspeptic symptoms. Stagnation in the proximal jejunal loop or duodenum should also be considered as a probable cause of dyspepsia[10] (afferent loop stasis with reflux; Wells and Welbourn, 1952).

Schindler[7] observed "a terrifying number of patients" who after gastric surgery have become permanently incapacitated. Schindler centered his attention on gastritis from reflux as the cause of postoperative disability. Moderate or fleeting presence of biliary reflux and various degrees of gastritis are frequently found in patients with either the Billroth I or Billroth II methods of anastomosis. These patients to all appearances are well and satisfied with the results of surgery.

In the presence of a small gastric pouch the cardia frequently remains open, permitting reflux of duodenal contents into the esophagus. Although distress from reflux is frequent in patients after gastroenterostomy and gastroduodenostomy, greater distress should be anticipated with a similar quantity of reflux in a smaller than in a larger gastric pouch.

A. Afferent Loop Stasis with Reflux: This condition is caused by partial obstruction from kinks, tortions, or adhesions proximal to the gastroenteric stoma (Figs. 1*A*, *B* and *C*). Wells and Welbourn, in 1951, recognized malfunction from stasis in the proximal enteric segment and designated the syndrome as "afferent loop stasis with reflux."[16] In the retrocolic technique, stasis is commonly caused by high attachment of the afferent jejunal loop on the lesser curvature near the esophagus (Fig. 1*A*).

Case 2 (C15972EH).—E. P., housewife and nurse, aged 34, underwent a subtotal gastrectomy in 1946 by the Hofmeister-Finsterer retrocolic technique for duodenal and gastric ulcers. Soon after a meal she began to complain of pain under the right costal margin and in the right shoulder blade accompanied by nausea and vomiting of bile. She became jittery and flushed and could feel and hear her heart beat. She was unable to tolerate certain foods. Her stools were constantly loose and clay-colored. She lost about 25 lb. and was unable to engage in her nursing profession or attend to her housework. On Feb. 12, 1949, I converted the retrocolic Hofmeister-Finsterer anastomosis into a retrocolic pantaloon anastomosis. The patient gained more than 20 lb. after the operation. All her symptoms disappeared. She has full-time employment in a local hospital in a psychiatric ward and attends to her household duties. She has gained 25 lb. in weight.

Comment.—Symptoms in this patient were indicative of an afferent loop stasis with reflux and with dumping mechanism of malfunction.

Case 12 (C41415EH).—J. W., a physician, aged 38, underwent two gastrectomies after 1946. Later he was operated on for intestinal obstruction. Because of bilious vomiting and enteroenteroanastomosis was done in 1949. Nevertheless, he was not relieved of his nausea and still complained of bilious vomiting, which he experienced about once weekly. If the vomiting came immediately after eating, it usually consisted of food. There was a continual feeling of nausea. Perspiration with an increased pulse rate and extreme weakness like that of a "mild form of shock" usually came during or immediately after mealtime. Relief was obtained after the patient lay down. He also had attacks of weakness which he ascribed to hypoglycemia. He lost 67 lb. from his preoperative weight of 175 lb. He was unable to attend to his professional duties. The patient was explored on Dec. 16, 1950. A minimal gastric pouch was found. The antecolic Polya anastomosis was converted into an antecolic pantaloon anastomosis.

The patient reported that he "benefited wonderfully well by the operation." In a late report he stated that his weight had risen to 140 lb. from a minimal weight of 108 lb. He is well satisfied with the results of the operation and is busily engaged in the practice of medicine. He is still unable to tolerate soups and milkshakes and finds it necessary to lie down after meals.

Comment.—It is apparent that the patient's symptoms indicated the whole gamut of postanastomotic and postgastrectomy mechanisms of malfunction, with afferent loop stasis and reflux as the predominating cause of his disability (Fig. 2*A* and *B*).

Case 19 (C49793EH).—A. M. B., a female school teacher, aged 67, underwent an appendectomy, cholecystectomy, and pyloroplasty in 1937. In 1950 she was subjected to a gastrectomy because of symptoms due to obstruction. In a letter she stated that she was told by her surgeon that she would always have bile come through her stomach. "All the bile must come through my stomach now." She was unable to tolerate many foods and vomited bile two or three times daily. She would get up at

night, take a cup of water, press on her abdomen and drain the bile. She lost 45 lb. The patient underwent an exploratory operation on July 1, 1951. I found a retrocolic anastomosis with the mesocolon sutured to the jejunal loops. The proximal jejunal loop was kinked with some torsion about 15 cm. proximal to the lesser curvature of the gastric pouch. The new gastrojejunal continuity was reestablished retrocolic by the pantaloon technique No. 1.

Excerpts from the second letter which I received after I performed the pantaloon operation are inserted because these portray a history of an afferent loop stasis with reflux malfuction, as related by the patient herself and without any interrogation or prompting: "The operation did not do what we had hoped. It is a year of dragging, miserable existence. It is a terrible disappointment of finding that the thing it was to do for me is still not done. I am up three or four times in the night to throw up about a pint of clear bile fluid. When I get up in the morning I drain my stomach of yellow, liquid bile like this. (She inserted a line with a light yellow-colored pencil.) Sometimes a little darker. I think all the bile kicks back into my stomach. I sometimes wonder if there is a restriction of the tubes where they empty into the intestines. I can feel it labor often before it kicks back into the stomach. The bile builds up, and there is a warning for several hours before the bile comes. I buy Seven-Up by the case. That year of dragging, weak and almost helpless is something too rugged to face without apprehension." I performed surgery for the second time on May 26, 1953, and decided on a Roux-Y anastomosis distal to the jejunojejunal pouch. This patient made an excellent recovery and gained 17 lb. in six weeks before she went home. After the Roux loop anastomosis she wrote me: "I neglected writing until I could tell you that I was entirely well as I was in my teens. Well I can say this, and very gratefully, I am much stronger than I have been in five years. I do not tire easily. I go from daylight until 9 or 10 at night often without thinking to rest. No bile ever; and I can eat with the rest of them."

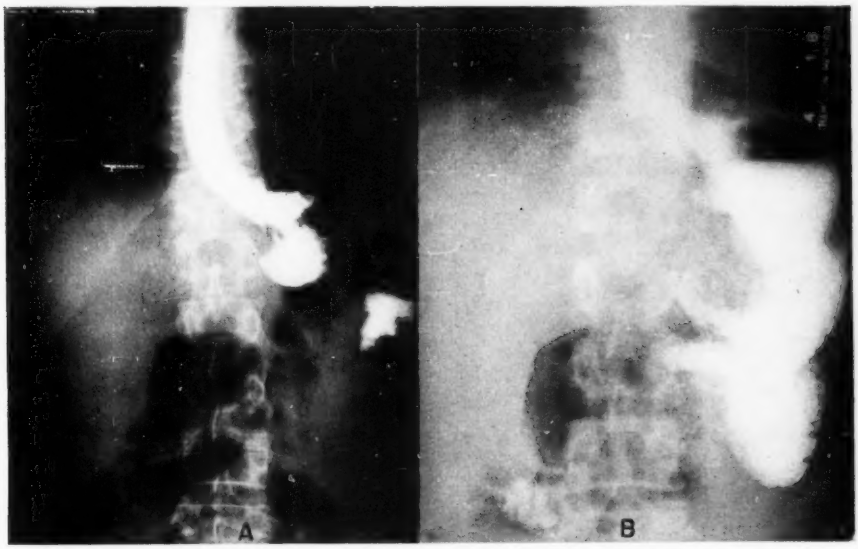


Fig. 2 (Case 12).—*A*, roentgenogram after an antecolic gastrectomy with a minimal gastric pouch. Lower shadow corresponds to the enteroenteroanastomosis which failed to forestall reflux. Afferent loop stasis and possibly also efferent loop stasis. *B*, roentgenogram sent in by the patient. Only a minimal gastric pouch. Abundant capacity of the jejunojejunal pouch with a *Magenblase*.

Comment.—It is clear that the crippling and unrelenting postgastrectomy disabilities in this patient were manifestations of an afferent loop stasis with reflux. The weakness and fatigue in this patient before the duodenal contents were side-tracked apparently resulted from the loss of electrolytes through vomiting.

Case 22 (S. P. H.).—A. A. A., a man, aged 27, was operated on for a perforated ulcer in 1948. He underwent a gastroenterostomy and vagotomy in 1949. After repeated hospitalization he was subjected to a gastrectomy in 1950. He continued to be

annoyed by nausea, heartburn, and bilious vomiting. He vomited more than a cupful of blood on numerous occasions. The relief from nausea was obtained by forceful vomiting. He was hospitalized subsequently several times and treated by continuous gastric lavage and intravenous infusions. He complained of weakness, light-headedness, nervousness, and perspiration and also severe abdominal cramps. He lost approximately 20 lb. in one year.

I performed an exploratory operation on this patient on Dec. 13, 1951. The anastomosis was found antecolic with an extremely wide stoma extending from the cardia on the lesser curvature side to the greater curvature side for a distance of about

Fig. 3 (Case 22).—Extremely long stoma with an antecolic anastomosis. Afferent loop kink from high attachment on the lesser curvature. Absence of barium shadow in the afferent loop.

12 cm. The afferent jejunal loop was kinked from high attachment near the cardia. There was no evidence of ulceration. The gastrojejunal continuity was reestablished retrocolic by the pantaloon technique.

The patient made an uneventful recovery, has had no vomiting, and has gained 9 lb.

Comment.—This patient's symptoms were indicative of an afferent loop stasis with reflux. The absence of a barium shadow in the afferent loop which was noted in this patient's roentgenogram is corroborative evidence (Fig. 3). Since no ulceration was found during the exploratory operation and since there was an absence of acid after histamine stimulation, it may be assumed that the repeated hemorrhages were due to gastritis from tryptic digestion.

Case 25 (359117EH).—C. N., a physician, aged 52, underwent a gastroenterostomy, a pyloroplasty, and a Hofmeister-Finsterer retrocolic gastrectomy on three antecedent and separate occasions. After the gastrectomy he became "wretched" because of continuous nausea, with infrequent bilious vomiting and a number of other complains. He was unable to attend to his professional duties. Exploration on Feb. 21, 1952, disclosed a kink in the proximal jejunal segment where the previous jejunal stoma had been closed during the dismantling of the old gastroenterostomy. I converted the previous anastomosis of the Hofmeister-Finsterer type to a retrocolic pantaloon method No. 1.

The patient wrote: "Really, it is so wonderful to feel human again." He also resumed his professional duties soon after the corrective surgery.

Comment.—Symptoms in this patient, together with the absence of barium shadow in the duodenum, suggest that malfunction was due to an afferent loop stasis with reflux.

B. Afferent Loop Stasis Without Reflux: An elderly patient with bilious vomiting after gastroenterostomy was relieved of his vomiting after the pantaloon anastomosis. He still complains of nausea. It may be assumed that in this patient, as in a few others, the cause of nausea may well be an afferent loop stasis without reflux.

C. Pendulum Reflux: Certain types of gastrojejunal anastomoses favor the entrance of ingested foodstuffs into the proximal enteric segment by gravity. Such placement of a gastrojejunal stoma, particularly when it is wide, also favors the entrance of biliary and pancreatic secretions into the gastric pouch (Fig. 1*D* and 1*E*). One can follow the seesaw movements of a barium swallow between the proximal intestine and the gastric pouch. Roentgenograms depicting an antecolic Polya gastrectomy with the jejunal loop directed from left to right frequently show a heavier shadow in the proximal jejunal loop (Fig. 4*A*). The presence of a barium shadow in the afferent enteric loop is not necessarily indicative of pendulum reflux malfunction unless the symptoms war-

Fig. 4 (Case 3).—*A*, typical roentgenogram of an antecolic Polya gastrectomy with the afferent loop placed at the greater curvature side. The shadow in the afferent jejunal loop is more prominent than in the efferent loop. Although this method of anastomosis is widely employed, it resulted in alarmingly grave disabilities from a pendulum mechanism of reflux. *B*, roentgenogram after the pantaloon anastomosis. This patient gained 50 lb.

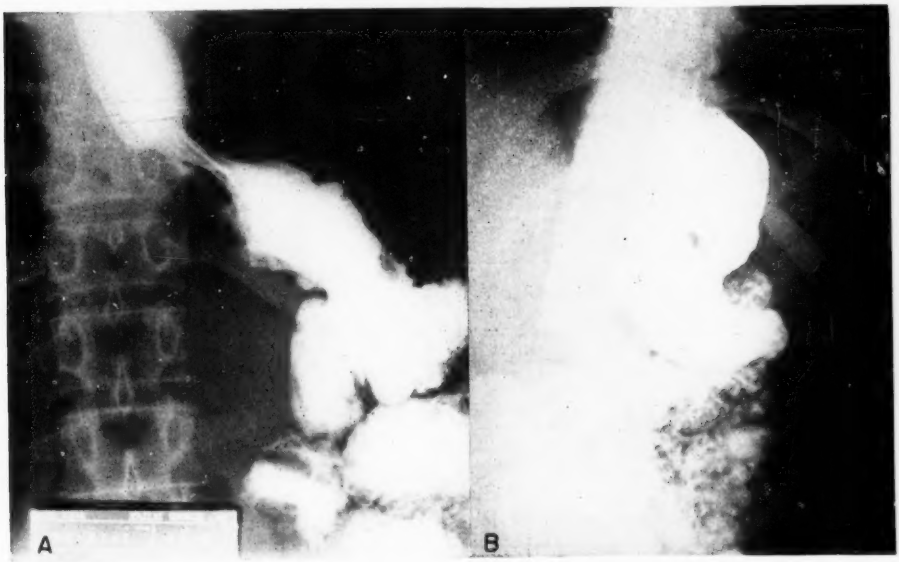
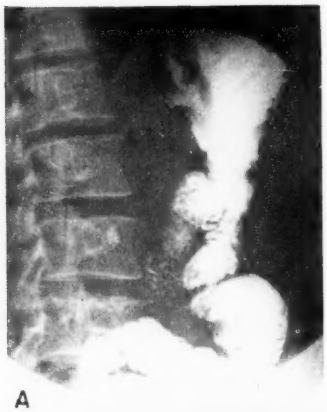


Fig. 5 (Case 8).—*A*, roentgenogram of a retrocolic Hofmeister-Finsterer anastomosis with reflux either on the basis of iso-peristaltic or pendulum mechanisms. *B*, roentgenogram taken after a pantaloon anastomosis. Barium shadow in the jejunojejunal pouch equals that of the remaining gastric pouch. Valve mechanism is visible.



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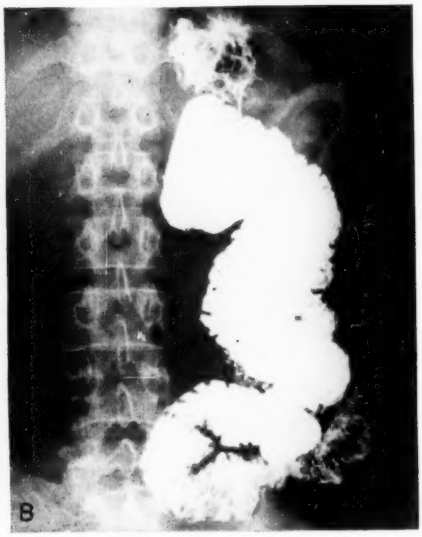


B

Fig. 6 (Case 1).—*A*, roentgenogram of a conventional Hofmeister-Finsterer retrocolic gastrectomy with no apparent roentgenologic evidence of malfunction. Grave and intractable postgastrectomy side-effects from reflux and dumping. *B*, postoperative roentgenogram after the pantaloon anastomosis. Only a minimal barium shadow is seen in the small gastric pouch. Large jejunojejunal pouch. Patient made complete rehabilitation.

rant such a diagnosis. The pendulum mechanism of reflux was responsible for serious symptoms from vomiting and anorexia in several of our patients.

Case histories of patients with either pendulum reflux or isoperistaltic reflux as the predominating cause of malfunction.

Case 1 (BD6559EH).—E. L., a housewife, aged 45, was subjected to a conventional retrocolic gastrectomy by the Hofmeister-Finsterer technique during August, 1947. She complained of being weak, tired, with a nauseating taste in her mouth, continual belching, spitting up of bile, and distress in the epigastrium after eating. She either had frequent episodes of diarrhea or was constipated. She lost 20 lb. On Oct. 26, 1948, the antecedent anastomosis was dismantled and a retrocolic pantaloon anastomosis No. 1 made. The patient now manages to work in her garden, attends to her household duties, and maintains full-time employment in a retail establishment. She has no more pain after eating, no belching, and no spitting up of bile. She tolerates all foods and has gained 36 lb.

Comment.—Symptoms were indicative of an uncomplicated isoperistaltic reflux with dumping. It may be assumed that the pantaloon anastomosis relieved also dumping (Fig. 6*A* and *B*).

Case 3 (C18595EH).—D. R., a farmer, aged 26, was subjected to an antecolic Polya gastrectomy four months before I was consulted. He complained of persisting vomiting of bile, hiccup, and anorexia. He lost 47 lb. in four months and was unable to work. I performed a retrocolic pantaloon anastomosis on April 4, 1949. The patient made an excellent recovery; he gained 50 lb. and resumed his farm work.

Comment.—Figure 4*A* demonstrates a roentgenogram of a Reichle-Polya antecolic anastomosis with the jejunal loop directed from left to right. It is to be noted that the barium shadow is somewhat heavier in the afferent jejunal loop. This patient was hopelessly disabled because of loss of bile and pancreatic juice and anorexia. This is clearly a pendulum mechanism of reflux malfunction. In spite of precipitous emptying of the barium swallow after the antecedent Polya and after the pantaloon anastomosis, there were no symptoms indicative of dumping malfunction (Fig. 4*A* and *B*).

Case 8 (C36096EH).—E. T., a farmer, aged 26, was subjected to a gastrectomy for a bleeding duodenal ulcer in 1946 and later to a vagotomy and removal of an additional part of the remaining stomach because of a hemorrhage from a jejunal ulcer.

The patient began to complain of precordial pain and distress and also pain between the shoulder blades with frequent vomiting of bile and food. He was unable to sleep in a reclining position and

awoke two or three times during the night to bring up half a cupful of bile. He usually drank about 30 cups of tea during the day and 10 cups after awakening before he could tolerate any food. He lost about 40 lb. The antecedent retrocolic Hofmeister-Finsterer gastrectomy was dismantled on Aug. 3, 1950, and a retrocolic pantaloon anastomosis by the No. 1 technique made. The patient made an excellent recovery. He is now able to sleep in a reclining position. He has gained 34 lb. and tolerates all foods, although he is unable to eat large meals. He works long hours in the lumber industry.

Comment.—The roentgenogram after the Hofmeister-Finsterer anastomosis demonstrates a minimal gastric pouch and good functioning of the afferent and efferent loops which fill and empty normally (Fig. 5*A*). The precordial pain and distress and the pain between the shoulder blades which disappeared after the pantaloon anastomosis, may be explained on the basis of esophagitis from reflux of pancreatic juice and bile. This is clearly an uncomplicated isoperistaltic or pendulum mechanism of reflux which followed a retrocolic Hofmeister-Finsterer gastrectomy (Fig. 5*A* and *B*).

The symptoms and disabilities because of reflux vary in character and intensity. Pain in the right hypochondrium and in the back and a feeling of pressure and nausea are symptomatic of the afferent loop stasis mechanism with or without reflux. Nausea results from the distention of a semiclosed afferent loop or from its motor activity initiated by the passage of food into the efferent enteric loops. Some patients have stated that they eat a substantial meal and vomit about a half to a glassful or unbelievably large amounts of pure bile within 10 to 20 minutes. Relief is immediate after vomiting. In the presence of any reflux mechanism when the gastric pouch is minimal, bile-stained vomitus mixed with food frequently comes shortly after a meal. Vomiting of food mixed with bile is also induced by overdistention of the gastric pouch (gastric pouch malfunction). Others complain of a bitter taste in the mouth, sore throat, and inability to sleep unless they assume a propped-up position. Presternal pain and discomfort may be explained on the basis of esophagitis from reflux of the duodenal contents. Clay-colored stools, steatorrhea, and diarrhea occurred in several of our patients from vomiting of copious amounts of bile-stained fluid. Diarrhea frequently occurred after a large vomitus of bile. In several patients the feeling of fatigue and lassitude completely disappeared after they ceased losing electrolytes through vomiting.

The case history of a patient demonstrates motor malfunction and reflux after a Haberer-Finney method of gastrectomy.

CASE 13 (C39511EH).—J. T., a laborer, aged 68, was subjected to a gastrectomy with a Haberer-Finney end-to-side anastomosis of the Billroth I method for carcinoma on November, 1950. He was able to tolerate only very small quantities of food and complained of severe presternal discomfort and burning. He lost 20 lb.

I took down the gastroduodenostomy on Feb. 21, 1951, and made an antecolic gastrojejunojejunal anastomosis by the pantaloon technique No. 1. The patient made a remarkable recovery, tolerated all foods, and gained 43 lb. He died 26 months later from carcinomatosis.

Comment.—This illustrates motor malfunction after a Finney-Haberer gastrectomy in spite of an unusually wide stoma and also esophagitis from reflux (Fig. 7*A* and *B*).

APPRAISAL OF THE PANTALOON ANASTOMOSIS AS THE INITIAL OPERATION FOR BENIGN AND MALIGNANT LESIONS AND AS A CORRECTIVE OPERATION FOR POST-ANASTOMOTIC AND POSTGASTRECTOMY MALFUNCTION

The gastrojejunojejunal anastomosis was introduced by Steinberg and Starr in 1934 (Fig. 8*A*).[9] Hoag and Saunders [4] employed a similar operation in 1939 for the relief of acute stomal obstruction after gastrojejunal anastomoses. An improved variation of the original gastrojejunojejunal anastomosis was employed for the first time in 1943 with the intention of preventing a possible stenosis (Fig. 8*B* and *C*). It was not until 1946 that a decision was made to explore this operation with the express purpose of forestalling reflux. The completed operation resembles a pantaloon (Fig. 8*B* and *C*). Tech-

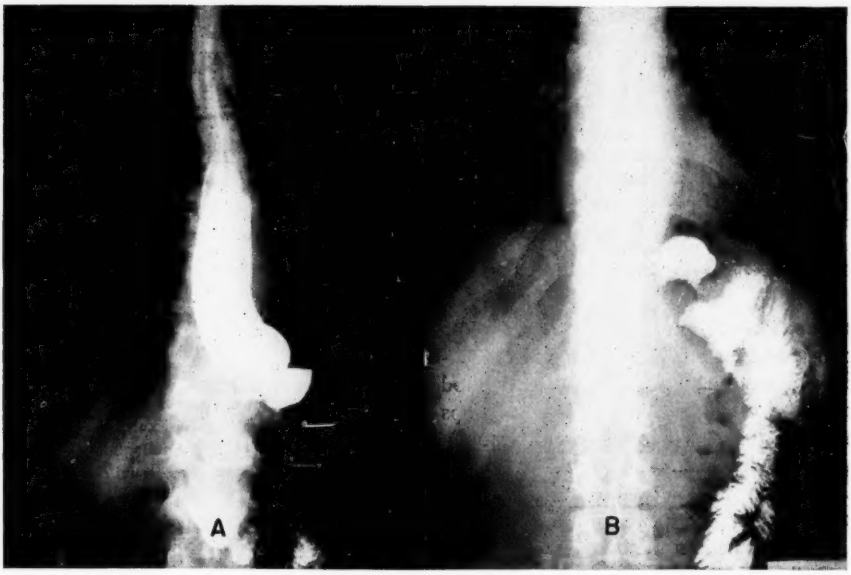


Fig. 7 (Case 13).—*A*, roentgenogram after a Billroth I Haberer-Finney gastrectomy, which resulted in motor malfunction with reflux. *B*, roentgenogram after an antecolic pantaloon anastomosis. Minimal gastric pouch is visible. Good function. Patient gained 43 lb.

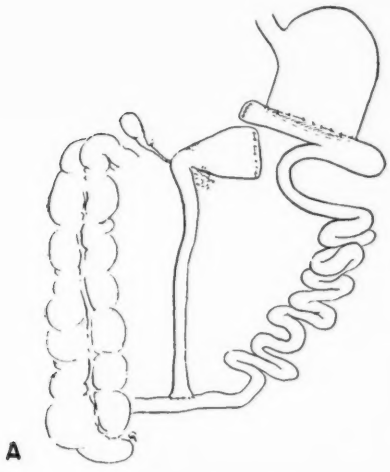
nical details and physiological implications of this operation were presented in a preliminary communication.[11] The pantaloon anastomosis has now been employed 260 times with partial gastrectomies as a primary operation for benign lesions, 25 times with partial gastrectomies as a primary operation for malignant lesions, 12 times with total gastrectomies, and in 33 patients for the relief of disabling postanastomotic and postgastrectomy side-effects. Since 1924, a total of 562 patients with benign lesions were subjected to a Hofmeister-Finsterer anastomosis and 29 patients, also with benign lesions, to a Billroth I anastomosis. Thus far, no perceptible differences have been noted as between patients with the conventional methods of anastomosis and those with the pantaloon anastomosis in relation to the incidence and degree of such disturbances as (*a*) ability of a number of patients to partake of large meals, (*b*) intolerance to concentrated sugars, and (*c*) incidence of postoperative weight loss. Both of these operations, the retrocolic Hofmeister-Finsterer technique and the pantaloon method, relieved the patients of the original ulcer distress and gave satisfactory functional results in more than 90% of the cases. Of the 562 patients with a Hofmeister-Finsterer type gastrectomy, 6 were personally subjected to corrective surgery because of malfunction. Of the 29 patients with a Billroth I gastrectomy, 2 were personally subjected to corrective surgery for reflux malfunction. The known incidence of jejunal ulcer is about 1% after both the conventional and the pantaloon anastomosis. Nevertheless, the more accurate assessment of the pantaloon anastomosis in relation to the incidence of jejunal ulcers must await a longer period of observation.[13] Thus far, there has been no occasion to reoperate for malfunction on any of the 298 patients with primary gastrectomies by the pantaloon technique. A middle-aged woman with a primary pantaloon gastrectomy, emotionally disturbed and with a history of chronic bowel distress, bitterly complains of pain in the right hypochondrium and also of a multiplicity of other symptoms. It is now apparent that the gastrectomy was ill-advised because of the presence of only

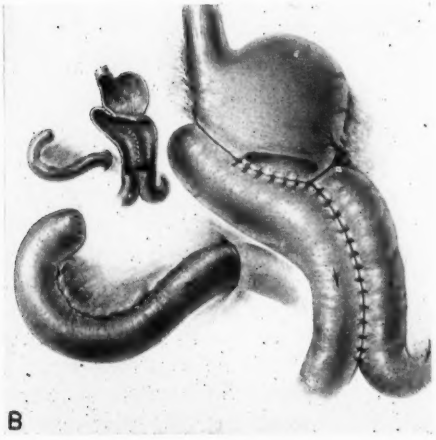
a minimal duodenal scar. The nature of her epigastric pain and distress is still undetermined.

It should be made clear that the Hofmeister retrocolic gastrectomy, popularized by Finsterer, has given lastingly good results. In my opinion, failures after the Hofmeister-Finsterer anastomosis usually result because of deviations from the technique perfected and standardized by Finsterer and also from excessive sacrifice of gastric tissue, which was largely inspired by a faulty interpretation of the results of animal experiments. A two-thirds to three-fourths resection is usually sufficient to prevent a jejunal ulcer.

The success of the pantaloon anastomosis will depend on the added time and diligence commensurate with an enlarged technique. Unless the surgeon is experienced in gastric surgery, he should approach the pantaloon anastomosis with restraint. The pantaloon anastomosis has obvious advantages in operations for partial and total removal of the stomach, for cancers, and for gastric ulcers located near the esophagus. The operation permits a generous overlapping of jejunal tissue around the stoma with less danger of stenosis or leakage than in the conventional methods. In total gastrectomies or in the presence of only a minimal gastric pouch, the addition of a generous jejunal pouch assures better functioning and prevents reflux.

The pantaloon anastomosis proved effective in relieving crippling postanastomotic disabilities in a number of patients. The 33 patients who were personally subjected to 39 corrective procedures for malfunctions had previously received 68 miscellaneous operations either for the initial lesion or for





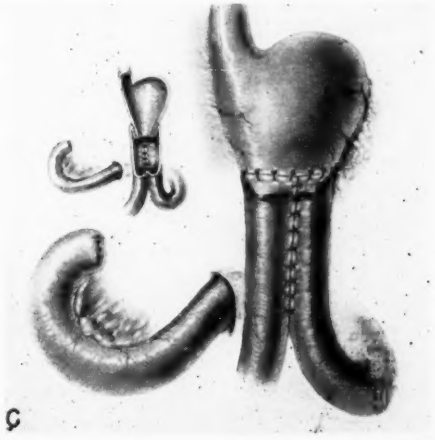


Fig. 8.—*A*, diagram of the original gastrojejunojejunoplasty presented by Steinberg and Starr [9] in 1934. *B*, the first technique of the pantaloon anastomosis which was employed in the greatest number of primary resections. *C*, the second technique of the pantaloon anastomosis is more exacting than the first method. The second technique is preferred when possible in reoperations for malfunction or for jejunal ulcer. In reoperations the jejunal loops are found wide, making this anastomosis feasible.

subsequent failures. Sixty of the antecedent operations which resulted in failure were done elsewhere. There were 37 antecedent gastrectomies of various types and 13 antecedent gastroenterostomies, also 4 vagotomies and 4 pyloroplasties. It is interesting to note that the only three gastrectomized patients who also received an enteroenteroanastomosis, either with the initial operation or later as therapeutic measure to prevent reflux, were not relieved of this form of malfunction (Fig. 2*A*, Case 12).

The most frequent and troublesome complaint in these 33 patients with postanastomotic and postgastrectomy malfunctions were bilious vomiting with tasting of bile and nausea. Only about eight patients vomited food. Upper abdominal pain and distress, occasionally pointing to the right hypochondrium, right shoulder blade (afferent loop stasis) or presternal area (esophagitis), were prominent symptoms in 15 patients. Bowel colic, pale and diarrheal stools with steatorrhea, rarely altering with constipation, were encountered in 11 patients. Intolerance to milk and pastries, with early postprandial symptoms such as weakness, lassitude, perspiration and belching which forced the patients to assume a recumbent position, were not so frequent as nausea and bilious vomiting. The history of hypoglycemic attacks, relieved by food, were obtained in eight patients. A number of patients were greatly emaciated, haggard, and depressed, complaining of weakness and fatigue. Seven became addicted to narcotics. Twenty-four were unable either to earn a livelihood or to attend to their housework. The average weight loss recorded in 24 patients was 30 lb. The minimal loss was 12 lb., and the maximum 67 lb. Five patients experienced repeated hemorrhages with no demonstrable ulcer to account for them. A number had repeated hospitalizations. One patient was continuously hospitalized for one year.

Twenty-five patients were relieved of the distressing symptoms after reparative surgical procedures with either significant recovery or with marked or fair improvement. These patients were able to return to their previous occupations. In some patients symptoms apparently resulting from dumping were either ameliorated or disappeared altogether. There was still some intolerance to concentrated sugars, milk, or other foods in a small number of patients. Such postprandial symptoms as sweating, lassitude, and palpitation, which previously compelled these patients to lie down or "flop down," either disappeared completely or were mitigated. All the patients with pale and diarrheal stools were relieved of these symptoms. Some became constipated. The average gain in weight recorded in 19 of the rehabilitated group was 22 lb.; the minimal gain was 4 lb. and the maximum, 50 lb. Of this group of 25 patients who were relieved of their disabilities, there were 3 who were not relieved until the addition of a Roux loop anastomosis was placed distal to the jejunojejunal sacculation (See Case 19).

In seven patients the results are considered questionable or a total failure. Some failures were due to diverse circumstances such as domestic difficulties, psychoneurosis, loss of a major part of the small intestines, antecedent vagotomy and above all, ill-advised selection of patients for corrective surgery.

In two cases in which the operation failed, the indications for the primary operations were not proved. The case histories revealed severe emotional disturbances in both patients with a multiplicity of chronic bowel complaints unrelated to ulcer distress. A 55-year old housewife who became an invalid after four antecedent operations died of shock after a Roux-Y loop anastomosis. The other patient, a 46-year-old housewife, became greatly emaciated after a minimal gastrectomy with a conventional anastomosis. Her condition worsened after the pantaloon anastomosis.

A drainage operation on the stomach in the absence of indications is certain to evoke severe manifestations from dumping and profoundly aggravate preexisting complaints. On the other hand, when indications for surgical interference because of intractable ulcer dis-

Data on Conventional Gastrectomies for Benign Lesions and on the Pantaloon Method of Gastrectomy for Benign and Malignant Lesions and for Postanastomotic Malfunction

Type of Operation	Patients, No.	Deaths, No.	Mortality, %
Gastrectomies by the conventional methods of anastomosis for benign lesions.	591	10	1.7
Gastrectomies by the pantaloon method of anastomosis for benign lesions.....	260	3	1.1
35 pantaloon gastrectomies and 4 Roux-Y loop anastomosis for malfunction..	33	2	5.3
Partial gastrectomies by the pantaloon method for cancer	25	1	4
Total gastrectomies by the pantaloon method for cancer	12	1	8.3
Total no. of patients....	892	15	1.7

The conventional methods of anastomosis for malignant disease have been discontinued since 1946. Otherwise these data include all operations for duodenal, jejunal, and gastric ulcers, also emergency operations for hemorrhage by both methods of anastomosis, and partial and total gastrectomies for malignant lesions by the pantaloon technique.

The first gastrectomy for a duodenal ulcer was done in 1924; the last gastroenterostomy was done in 1925.

There were no fatalities from a blow-out of the duodenal stump, although there were a number of duodenal fistulae.

tress or crippling postanastomotic side-effects were definite, the patients benefited from the operation in spite of unrelated bowel complaint and emotional disturbances. There are always unpredictable variations and technical imperfections inherent in any method of gastroenteric anastomosis which may influence the result of an operation.

Efforts to untangle the gastrectomy aftermath were frequently misdirected because attention was largely centered on dumping as the all-inclusive cause of malfunction. Results of corrective surgery proved of merit in the clarification of a number of issues involved in functional disuturbances after gastric operations. It is now possible to recognize some mechanisms of malfunction with a measure of accuracy and select patients for reparative surgery with reasonable hope of success. Such pessimistic attitudes as those expressed by Schindler,[7] who considers the treatment of the "torturing intractable symptoms" of the postoperative stomach because of reflux as a hopeless task, and those of the late A. Hedley Visick,[14] who regarded bilious vomiting after gastric surgery as the most crippling and incurable, is no longer justified. It is precisely such postoperative disabilities that are most amenable to cure by surgical intervention. Wells and McPhee[15] report that they have treated 14 postgastrectomy patients with afferent loop stasis with reflux by jejunoplasty, with immediate and satisfactory results. Johnson and Orr[5] report that the "vorse dumper" was relieved by a Steinberg jejunoplasty.

SUMMARY AND CONCLUSIONS

Issues involved in the postanastomotic and postgastrectomy side-effects become more understandable when interpreted on the basis of the following separate mechanisms of malfunction: (1) gastric pouch malfunction; (2) dumping malfunction (efferent loop syndrome); (3) reflux malfunction with afferent or efferent loop stasis.

Thirty-three patients who underwent 68 miscellaneous antecedent operations were later subjected to 39 corrective operations for a variety of disabling postanastomotic and postgastrectomy malfunctions. The average loss of weight recorded in 24 of these patients before corrective surgery was 30 lb.

Twenty-five of these patients obtained either significant or satisfactory results and were able to return to their previous occupations. The average gain in weight recorded in 19 patients after corrective surgery was 22 lb. In five patients the results are considered inconclusive and in two the operation was a failure.

Crippling side-effects because of the various mechanisms of reflux and enteric loop stasis were more frequent than crippling side-effects from dumping.

Postanastomotic reflux malfunction, with and without afferent or efferent loop stasis, can be recognized and rectified by definitive surgical methods. An enteroenteroanastomosis does not regularly prevent reflux.

The original gastrojejunojejunoplasty was introduced by Steinberg and Starr in 1934.[9] Since 1946, an improved method of gastro-

jejunojejunal anastomosis, designated as the pantaloon method, was employed in 330 patients for benign and malignant lesions and for postoperative malfunctions, with an overall mortality of 1.5%, as compared with 591 patients subjected to gastrectomy by the conventional methods of anastomosis for all benign lesions with a mortality of 1.7%. The end-results in patients with the conventional method of anastomosis and in those with the pantaloon anastomosis when employed as the initial operation were equally satisfactory.

Undesirable side-effects after the Hofmeister retrocolic anastomosis frequently result because of deviation from the technique standardized by Finsterer and also because of the injudicious removal of increasingly larger parts of gastric tissue dictated by faulty interpretation of the results of animal experimentation.

The pantaloon anastomosis has obvious technical and functional advantages over the conventional method in all primary operations when all or most of the stomach is sacrificed.

The pantaloon anastomosis proved of singular merit in relieving crippling postanastomotic side-effects.

The success of the pantaloon anastomosis will depend on the added time and diligence commensurate with an enlarged technique. Unless the surgeon is experienced in gastric surgery, he should approach the pantaloon operation with restraint.

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Newer Methods for Treatment of Urethral Stricture

R. H. FLOCKS, M.D.
LOUIS J. PRENDERGAST, M.D.
HANS MARBERGER, M.D.
and
DAVID CULP, M.D., Iowa City

Although stricture of the anterior urethra is becoming less frequent, owing to the relative disappearance of venereal disease, trauma, both external and internal, still produces a sufficient number of strictures of the anterior urethra to make these conditions fairly frequent. In addition, the greater incidence of operations upon the prostatic urethra has increased the number of instances of stricture formation at the bladder neck.

The majority of strictures of both the anterior and posterior urethra are adequately treated by conservative methods. These are sufficient to control infection and maintain a lumen which is satisfactory enough to prevent infection and back pressure effects upon the bladder and upper urinary tracts. Since these changes may be insidious, frequent check-ups by means of intravenous pyelography are necessary in addition to periodic study of the urethra and the urine.

Patients with strictures of the anterior urethra can usually be maintained satisfactorily by instrumental dilatation properly done once every few months with filiforms and Philips followers. Care must be taken not to overstretch the urethra and thus create new scar tissue. Occasionally, internal urethrotomy, with the use of an instrument such as the Riba electrotome, is necessary.

From the Department of Urology, The State University of Iowa.

However, a significant percentage of strictures of the anterior urethra cannot be treated by the methods just described, and a significant percentage of patients with bladder neck contracture are not cured by transurethral removal or suprapubic removal of the scar tissue at the bladder neck. New scar tissue forms, with further constriction, and the patient is in difficulty again. Newer methods for reconstruction of the anterior urethra and the prostatic urethra in these cases have recently been instituted which have produced satisfactory results. This has created a new outlook for patients with these conditions.

For the anterior urethra we have used at the University Hospitals for the past one and one-half years in a series of 25 patients the method of Johansen [1] of Sweden, which is an adaptation of the Denis-Browne * operation for hypospadias to urethral stricture. This operation for a stricture in the pendulous urethra is illustrated in Figures 1 to 5. A similar procedure is carried out for stricture of the bulbous urethra. The operation consists essentially of two stages.

The first stage creates a partial or complete hypospadias. This does two things: It institutes excellent drainage for the whole strictured area, in and about which recurring infections have taken place and puts the whole area at rest. It consists essentially of opening up the strictured area to the outside and suturing the remnants of mucosa to the skin area as illustrated in Figures 1 and 2.

At the second stage, utilizing the technique of Denis-Browne, the urethra is reconstructed, utilizing whatever remains of the mucosa and making up the remainder of the urethra from the surrounding skin edges as illustrated in Figures 3, 4, and 5. This is

* References 2, 3, and 4.

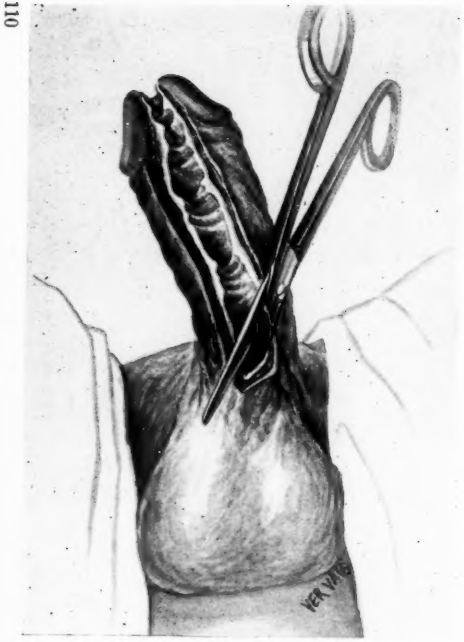


Fig. 1.— Johansen operation for urethral stricture. First stage. Stricture of pendulous urethra. The urethra is cut open widely, ventrally, to a point well above the stricture. The incision is carried right through the overlying skin.

Fig. 2.—Johansen operation for urethral stricture. First stage stricture of the pendulous portion. The mucosa of the strictured urethra is carefully sutured to the adjacent skin with interrupted 000 chromic absorbable (gut) sutures. The urine is side-tracked with an indwelling urethral catheter for six days. The patient then returns home for at least one month or longer if more time is needed for complete healing of first stage. Note that the urethral stricture area is at rest and is well drained.

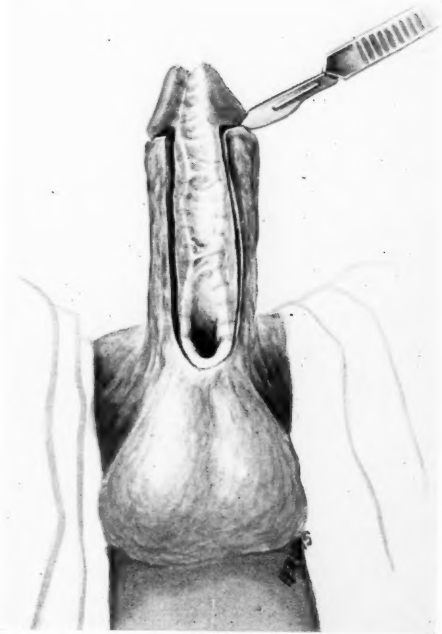


Fig. 3.—Johansen operation for urethral stricture. Second stage. Stricture of pendulous portion. An incision is made as illustrated leaving a wide portion of urethral mucosa and skin to be buried for the formation of the new urethra.

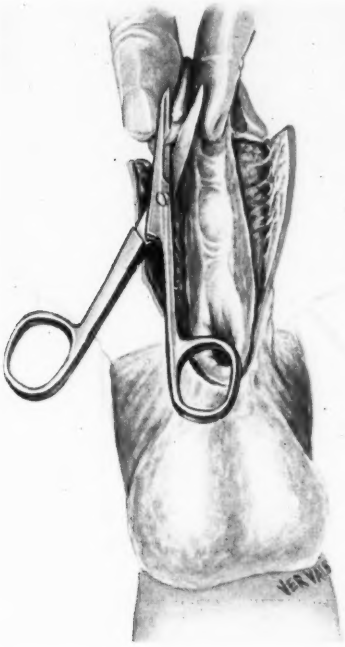
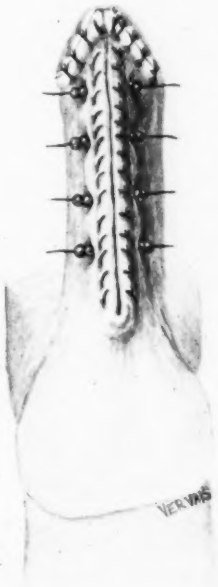


Fig. 4.—Johansen operation for urethral stricture. Second stage. Stricture of the pendulous portion. The lateral flaps are undermined widely as illustrated. If necessary a vertical incision for relaxation of tension may be made on the dorsum of the penis. This is usually a wise maneuver. The corona of the glans is denuded to make it possible to carry out the urethra to the tip of the glans before closure is carried out as illustrated in Figure 5. The urine is side-tracked by either a perineal urethrostomy or a suprapubic cystostomy.

associated with side-tracking of the urine by means of a perineal urethrostomy or a suprapubic cystostomy. The second stage is carried out approximately two months after the first procedure. This interval allows all of the surrounding infection to clear up. By carefully utilizing enough skin so that it will be easy to form a new urethra and utilizing the surrounding skin under just the right amount of tension, a successful urethra can be created in almost every case. The patient then will no longer require dilatation, will void very satisfactorily, and ordinarily will have no difficulty at all with hair formation because of metaplastic changes in the skin forming the new urethra. In our series of 25 patients results were uniformly good. An example of this is shown in Figure 6*A*, *B*, and *C*.

Bladder neck contracture following transurethral prostatic resection and other forms of prostatectomy can be very difficult to manage. It is hard to dilate these contractures because the dilating sounds and filiforms catch in the enlarged prostatic urethra since they cannot be readily directed through the tiny opening at the neck of the bladder which forms as a result of scar tissue. Transurethral resection of these bladder neck contractures is satisfactory in the majority of cases but in a very significant number, probably about one out of three, is followed by very rapid recurrence of the condition. Su-

Fig. 5.—Johansen operation for urethral stricture. Second stage. Stricture of the pendulous portion. Closure of lateral flaps. Care must be taken that no tension exists. Tension sutures of 32 steel wire with lead shot and beads are used. The skin edges are closed with interrupted 000 plain absorbable (gut) sutures. In about 10 days the side-tracking catheter can be removed, and the patient begins to void satisfactorily through the newly created urethra. Adequate chemotherapy in the postoperative period is of great importance.



for complete healing of first stage. Note that the urethral stricture area is at rest and is well drained.

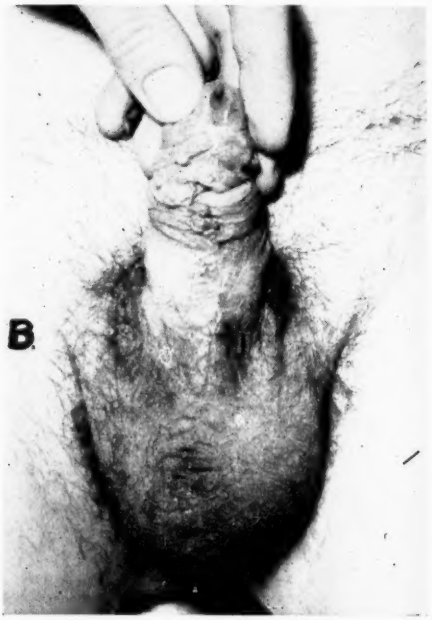


Fig. 6*A*.—Showing the appearance after the first stage has been completed. Note the excellent healing and the absence of induration.

Fig. 6*B*.—Appearance after second stage. Note new meatus. Note well-healed wound.

Fig. 6*C*.—Note urethral stream. Wide open urethra has been created.

prapubic removal of the scar tissue ordinarily is no more satisfactory than the transurethral technique but requires longer hospitalization.

In 1951, Young,† of San Francisco, described a new method of treatment for congenital bladder neck contracture which, instead of trying to accomplish the correction of the contracture by removal of scar tissue alone, added a very significant step to the procedure. This significant step was the enlargement of the bladder neck by the utilization of mucosa and musculature from the adjacent bladder wall. Thus a V-shaped tongue of bladder tissue was incorporated into the anterior portion of the prostatic urethra so that the entire prostatic urethra and bladder neck are markedly enlarged as a result of this additional flap of tissue. This, of course, is a very fundamental underlying principle in plastic surgery and worked very satisfactorily in children with congenital bladder neck contracture.

Reed Nesbit ‡ recognized the possibilities of this procedure for the treatment of acquired bladder neck strictures following prostatectomy of various sorts and utilized this first about two years ago in such a case with great success. We have utilized the

† References 5 and 6.

‡ Personal communication to the authors.

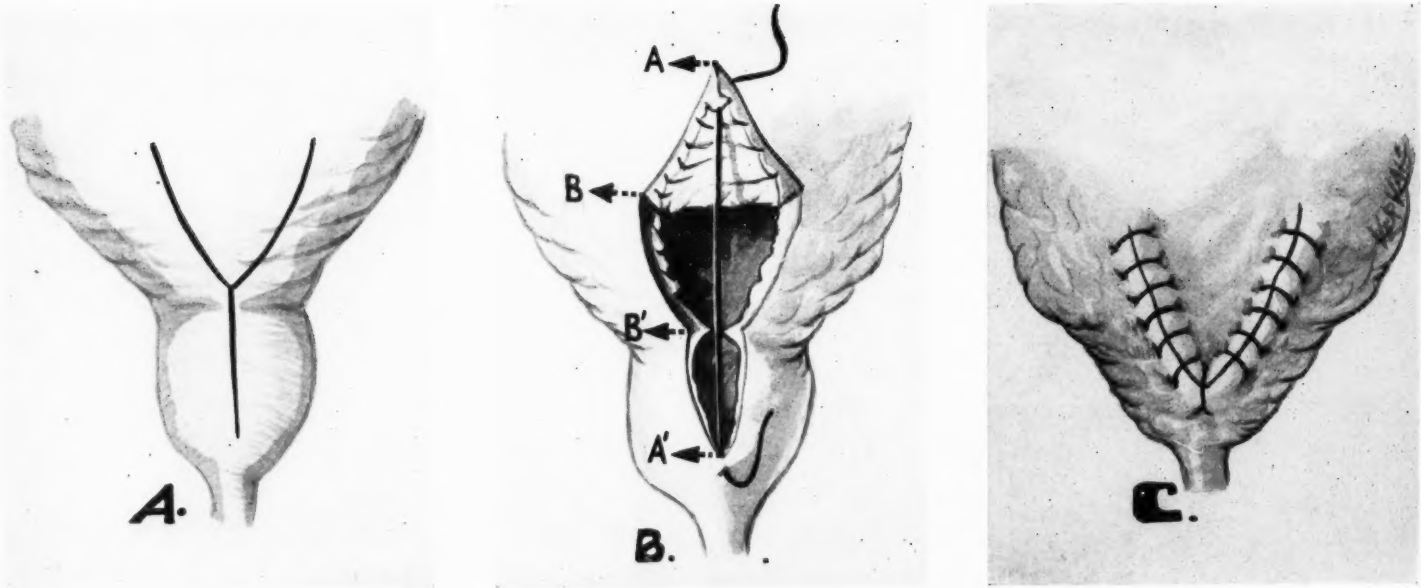


Fig. 7.—Plastic repair of contracture of bladder neck utilizing flap of adjacent bladder. *A*, Outline of incision. *B*, Incision achieved. Scar tissue removed. Ready to close with utilization of flap of adjacent bladder. *C*, Closure which has incorporated at least 2 cm. of bladder wall into bladder neck anteriorly.

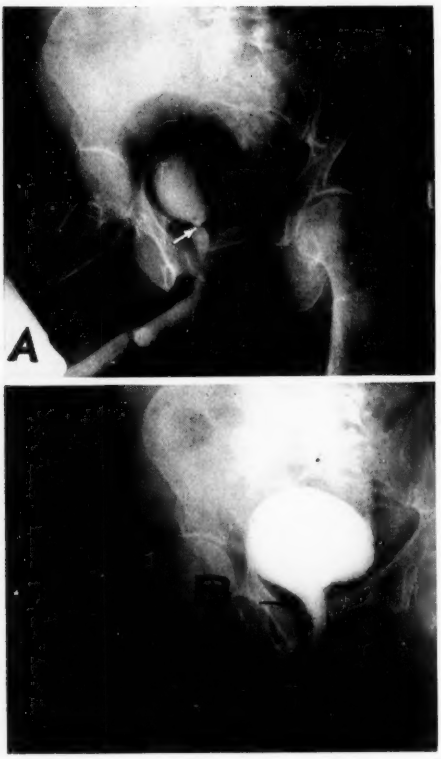


Fig. 8*A*.—Urethrogram of patient with bladder neck contracture after transurethral resection. Recurrent.

Fig. 8*B*.—Urethrogram after bladder flap. Note wide open bladder neck. Flap is actually visualized on the urethrogram.

same procedure, which is diagrammatically illustrated in Figure 7*A*, *B*, and *C*, in two cases with excellent results. Figure 8*A* and *B* are illustrative of one case.

SUMMARY

A brief discussion is given of the management of strictures of the anterior and posterior urethra of the acquired type, and a description of newer methods utilizing the fundamental principles of plastic surgery is given. For strictures of the anterior urethra the principles of Denis-Browne are used, and for strictures of the posterior urethra flaps of bladder wall are incorporated into the strictured area. The techniques with results in patients are illustrated.

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Coexistence of Renal Tumor and Solitary Cyst of the Kidney

Report of Two Cases

GERALD W. SCHWIEBINGER, M.D.
and
CLARENCE V. HODGES, M.D., Portland, Ore.

There is general agreement that surgical exploration of a solitary cyst of the kidney is indicated when other disease is suspected. There is not such agreement when other lesions cannot be demonstrated. What symptomatic and objective factors should be considered prior to recommending surgery in the patient with a solitary cyst of the kidney? Our recent experiences with two patients in whom renal tumor and cyst were independently present in the same kidney have prompted a reassessment of this problem.

DEFINITION

Cysts of the kidney have been classified into six general groups by White and Braunstein.[26] These are (1) congenital or developmental, (2) obstructive, (3) neoplastic, (4) vascular, (5) inflammatory, and (6) parasitic. Solitary cysts of the kidney fall into the congenital or developmental group. These are also referred to as simple cysts, serous cysts, and large solitary cysts, and, when bleeding occurs into them, hemorrhagic cysts. Some authors [23] differentiate the hemorrhagic cyst as a separate entity. It is with solitary cysts of the kidney that this paper is concerned. Such a cyst is defined by Herbut [15] as a large serous cyst located in any portion of the kidney and accompanied or unaccompanied by other large or small, primarily serous cysts. Grossly, these are whitish or bluish, thin, tense, translucent-walled structures containing a straw-colored fluid.

PATHOGENESIS

There are many theories of origin,[13] falling into congenital and acquired groups. Albarran felt that solitary cysts of the kidney were in reality a simple variety of polycystic degeneration confined to a restricted portion of the kidney. Kampmeier conjectured that they were the result of persistence and later expansion of cystic tubules which failed to collapse during fetal life. Willis and Virchow believed that occlusion of papillary ducts by inflammatory sclerosis was responsible. Hildebrandt postulated that failure of union of glomerular and tubular anlagen occurred with cyst formation. Hepler [14] found that he could experimentally produce cystic disease of the kidney by a combination of tubular blockage with localized ischemia. He further postulated that, since tubular blockage and ischemia may be produced by neoplastic growths of the kidney, a cyst secondary to a tumor would not be unusual.

SYMPTOMS

The symptomatology of a cyst of the kidney is primarily that resulting from local pressure, either upon the kidney itself or upon the neighboring organs. There is lack of agreement as to the incidence and the cause of hematuria in simple cysts. Gutierrez stated that hematuria with no other assignable cause is not infrequent. Shivers

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From the Department of Surgery, University of Oregon Medical School.

and Axilrod [22] recently reported three cases of solitary cysts, in one of which there was hematuria. Cahill [3] found, in 43 cases of solitary cyst of the kidney, only 6 had shown hematuria; all were ascribable to associated lesions. Greenberg, Brodny, and Robins [12] also felt that when hematuria was present it usually was due to a complicating factor. Prather [18] stated that even microscopic hematuria was infrequent. Braasch,[2] however, reported finding hematuria in 23 of 163 cases. In most instances a complicating factor was found. Of 15 patients with solitary cysts of the kidney operated upon at this institution, 7 were found to have had hematuria. One of these was secondary to a calculus and one to trauma, and two were associated with renal tumors. Hematuria was ascribed to the cyst in the remaining three cases.

PATHOLOGY

The more frequently associated lesions and complications from a solitary cyst of the kidney include calculi, hydronephrosis, pyelonephritis, torsion, trauma, and infection of the cyst contents. An uncommon finding is a tumor associated with this lesion. Scholl [21] reported a papillary cystadenoma in the base of a hemorrhagic cyst. Case 4 of Gutierrez's [13] exhaustive survey revealed an infiltrating Wilms' tumor in the wall of a cyst. Ritter [19] found a malignant hypernephroma arising from or associated with a cyst. Ezickson and Greene [8] reported a solitary cyst with adenocarcinoma in the wall of the cyst. LeComte [16] stated that a large proportion of hemorrhagic cysts have been found on painstaking histologic examination to contain carcinoma cells in their walls. Lowsley and Curtis [17] found a hypernephroma and, in another patient, a papillary cystadenoma associated with a cyst of the kidney. Colston [4] reported the occurrence of a solitary cyst and a papillary cystadenoma in the same kidney but unassociated as to location. Cunningham,[6] in 1915, described a case of cyst and carcinoma. Crabtree [5] found a solitary hemorrhagic cyst and a sarcoma in the same kidney. Greenberg's [12] Case 1 revealed a hypernephroma of the kidney with extension to the renal vein and an associated cyst of the lower pole. Rolnick [20] cites Quinby and Bright, who found a solid tumor associated with a solitary cyst of the kidney in three cases in a series of 36 solitary cysts. In Cahill's 43 cases one tumor was found adjacent to a cyst. Walsh [24] mentioned a case of hypernephroma associated with a solitary cyst in his discussion of the problem before the Royal Society of Medicine. Wheeler [25] cited a case of a cyst of the kidney, from which clear fluid was obtained by needle aspiration; on subsequent exploration it proved to contain an adenocarcinoma. Gibson [10] recently reported three cases of malignant growths associated with renal cystic disease.

REPORT OF CASES

CASE 1.—A 77-year-old white man, was admitted to the Urology Service with complaints of hematuria and dysuria of two months' duration. Physical examination was negative except for benign prostatic hypertrophy. Urinalysis revealed microscopic hematuria and pyuria. Cystoscopy revealed a granular, friable prostatic urethra with marked coarse trabeculation of the bladder. Retrograde pyelography revealed a large left hydronephrosis and a distorted poorly filled superior calyx. At operation a large, thin-walled, bluish solitary cyst was first encountered on the superior pole; on further dissection a small mass within the upper portion of the pelvis was felt. After nephrectomy, bisection of the kidney revealed this to be a papillary carcinoma arising from the superior calyx (Fig. 1*A* and *B*). Histologically this was identified as a papillary transitional cell carcinoma. A complete ureterectomy was subsequently performed and several papillary tumors of a similar histological nature were found within the urethral lumen.

CASE 2.—A 70-year-old white man, came to the Urology Service because of frequency, nocturia, difficulty in voiding, and dysuria. There were no complaints of hematuria or flank distress. Physical examination revealed an enlarged prostate but was otherwise not remarkable. Urinalyses on voided specimens revealed pyuria without hematuria. Retrograde pyelography revealed a space-occupying mass in the lower pole of the right kidney. The preoperative impression was a cyst of the right kidney. At operation a typical solitary cyst of the lower pole of the right kidney was found, but, in addition, on further mobilization of the kidney, a suspicious enlargement of the upper pole was noted. After nephrectomy bisection of the kidney revealed

Fig. 1*A*.—Surgical specimen superimposed on retrograde ureteropyelogram of left kidney, showing solitary cyst attached to superior pole with underlying, infiltrating papillary carcinoma. Distal ureter shows tumor implants.

Fig. 2*A*.—Right kidney showing solitary cyst of lower pole and independently arising adenocarcinoma of the upper pole.

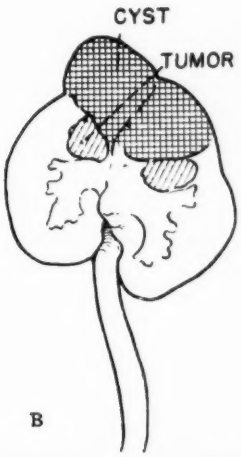

Fig. 1*B*.—Diagrammatic representation of left kidney.

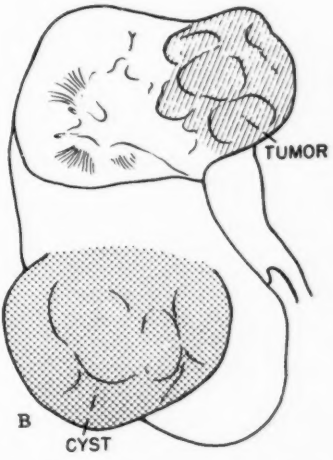

Fig. 2*B*.—Diagrammatic representation of right kidney.

a yellowish rounded mass, 4 cm. in its greatest diameter, in the cortex of the superior pole (Fig. 2*A* and *B*). Histologically this proved to be an adenocarcinoma.

DIAGNOSIS

The diagnosis of a tumor associated with a cyst may first be suggested by a history of hematuria in a patient with a cystic-appearing lesion of the kidney. Urethrocystoscopy and pyelography will give the answer in most cases. With the addition of renal angiography to our armamentarium, more diagnostic assistance may be had. In the specific case of solitary cysts with hematuria, aspiration of the cyst with injection of radiopaque medium as advocated by some authors * may give additional information, but it cannot be considered significant when no tumor is demonstrated and would have been fatally misleading in our cases. Cytological studies of the urinary sediment may be helpful but again are of little value when negative.

TREATMENT

The main problem arises when a solitary cyst is diagnosed and no obvious tumor is seen. Braasch, in discussing Lowsley and Curtis' paper,[17] said, "surgical exploration is necessary in practically every case, since the possibility of renal neoplasm must be excluded." Gutierrez concluded that the ideal course is to advise open surgical exploration of the kidney and the cyst by laparotomy with a view to resection of the cyst by one or another method. Herbut felt that the occurrence of a malignant tumor in conjunction with a serous cyst is frequent enough to warrant serious consideration in any given case. Prather, after an excellent review of the differential diagnosis between renal tumor and renal cyst, concluded that surgical inspection was still the most reliable method of differential diagnosis in most cases. Walsh, who agrees with Hepler in his postulation that tumor can be the cause of a solitary cyst of the kidney, excludes only the very small asymptomatic cyst from renal exploration. Ainsworth and Vest [1] on the basis of their experience dispute this method of management and rely on aspiration diagnosis of the solitary cyst. They further state that in 20 cases, apparently several with hematuria, they have aspirated clear fluid and felt that they have thus ruled out the coexistence of a renal tumor. The majority of the evidence and the collective opinions of the articles in the literature would indicate that this is a radical approach to the problem and not "conservative" management. The infrequency of the coexistence of two lesions in the kidney, one benign and one malignant, does not seem to us to be sufficient justification for such a gamble.

* References 1, 7, 9, and 11.

CONCLUSION

Two additional cases have been reported wherein the coexistence of a solitary cyst of the kidney and an independently arising tumor was noted. These reemphasized for us what the experience of others has noted previously, that solitary cyst of the kidney may be coexistent with more serious lesions. The cyst as the obvious lesion should not be construed as the only lesion present. When this finding is present with a clinical history of hematuria, more proof is required that the hematuria is due to cystic disease alone. It is evident, also, that a mere lack of hematuria is not a good reason for observing the lesion until such a symptom should develop. Admittedly the greater share of cysts operated upon will not have tumor either in their walls or in a separate segment of the kidney as in our two cases. However, we believe that surgical exploration is justified to exclude tumor while at the same time allowing appropriate treatment of the cyst.

SUMMARY

Two cases in which renal tumor and solitary cyst coexisted are reported.

Solitary cyst of the kidney justifies surgical exploration.

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Fractionation Technique in Translumbar Aortography and Femoral Arteriography

MARTIN LANGSAM, M.D.
ALEXANDER LEWITAN, M.D.
LOUIS NATHANSON, M.D., Brooklyn
and
DAVID WILENSKY, M.D.†

New surgical techniques in peripheral vascular diseases* have revived interest in arteriography. One of the basic problems is to visualize the aorta and the femoral artery in their entirety and in one procedure, in order to obtain information about their anatomical and functional state on several films.

Evidence of intimal narrowing or obstruction obtained in the usual procedure by using a single film does not permit a positive differential diagnosis between an organic and a spastic lesion. Multiple films taken in rapid succession are essential for proper diagnosis. For this purpose a special apparatus with rapid change-over of cassettes has been described.[3] However, such special apparatus is not readily adaptable to existing equipment and space.

Injection of radiopaque medium into the aorta, properly timed, will visualize the femoral arteries and eliminates the need of an additional open or percutaneous injection into the femoral artery. Satisfactory aortograms and femoral arteriograms can be obtained if the injection site into the aorta is kept below the renal arteries. Thus, the radiopaque medium is not wasted into a large visceral artery, and adequate visualization results.

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From the Peripheral Vascular and Radiological Sections of the Jewish Sanitarium and Hospital for Chronic Diseases.

† Dr. Wilensky died Aug. 30, 1954.

* References 1 and 2.

In the selection of patients referred for translumbar aortography, the following criteria were established:

1. No patient was accepted who had a history of acute coronary occlusion or cerebrovascular accident of less than three months' duration.

2. Patients with elevated values for blood urea or hypertensive patients with systolic pressure above 250 or diastolic pressure above 110 or with more than 1+ albumin in the urine were not acceptable.

3. Cardiac patients who were, in our opinion, not good risks were rejected.

4. For technical reasons, patients with flexion deformities of the lower extremities or spine could not be adapted to the flat surface of the x-ray table and, therefore, were not used.

Our procedure is as follows. We employ sodium acetrizoate (Urokon Sodium) 70% as the radiopaque medium. During the preceding day, an intravenous test dose is administered, consisting of 2 cc. of 35% sodium acetrizoate. If no undue reaction occurs, aortography is carried out the following day with local anesthesia, supplemented with trichloroethylene (Trilene). The patient is prepared with Pentobarbital (Nembutal) and 50 mg. of tripelennamine (Pyribenzamine).

Prior to the aortography, a flat film of the abdomen is taken with a metallic marker which is placed 7.5 cm. from the midline and 3.5 cm. from the costal margin. After the film is developed, a site of puncture is chosen that will permit placement of the needles below the level of the renal arteries, i. e., below the lower border of the first lumbar vertebra.

The needles are then inserted into the aorta; 5 cc. of sodium acetrizoate is injected, and a scout film is taken. This serves a dual purpose. First, the position of the needles is checked to see if they are placed at the correct level and not in one of the renal arteries. Thus, damage to the renal parenchyma is avoided.† If the puncture site is above the level of the renal arteries, the needles are removed and placed at a lower level, with the bevels directed cephalad. This is recommended to avoid unequal distribution of the radiopaque medium in the iliac arteries. The second purpose of this injection is to determine whether the patient will tolerate the procedure. In one case, we noticed vascular collapse after the injection of 5 cc. of sodium acetrizoate, although the injection site into the aorta was below the renal arteries. We promptly discontinued the procedure at this point. We believe that if we had used a larger dose in this patient, there might have been severer reaction.

If the needles are properly placed, the first injection of 5 cc. outlines the lower aorta as well as the bifurcation of the iliac vessels. The scout film is purposely overexposed to enable it to be developed more rapidly.

While waiting for the film to be developed, it is important to notice the return of a pulsating column of blood in the polyethylene tubing. If not, it is necessary to determine whether the needles are in the aorta and to watch carefully the condition of the patient. On one occasion, the failure of the return of the pulsating blood column was the first indication that the patient was going into vascular collapse. In over 50 aortograms there has been no difficulty with clotting in the needles or the polyethylene tubing.

For the second injection, 15 cc. of radiopaque medium is used. At this time, a regular Bucky film is taken which is large enough to cover the site of injection to the upper 2 in. of the superficial femoral vessel (Fig. 1). We obtain, in this fashion, two films showing various degrees of filling of the aorta and iliac vessels. Radiographically, the constancy of any particular filling defect can be assessed. On the second film, opacification of the iliac arteries is usually noted as well as the common femoral and its branches.

Fig. 1.—Aortogram.

Fig. 2.—Femoral Arteriogram.

† References 4 and 5.

Since approximately five minutes elapsed between the first and second injection, an excretion pyelogram must be noted (Fig. 1). If such findings are not present, a third injection is delayed until definite excretion by the kidneys can be observed. Each injection of the dye is carried out with the aid of an automatic apparatus.[6] X-ray exposures are timed to coincide with the injection of the last of the opaque medium.

If the procedure has progressed satisfactorily to this point, a third and final injection is made consisting of 35 cc. of sodium acetrizoate. Prior to this last injection, the patient is positioned on a tunnel which holds two 14×36 cassettes, each containing two 14×17 films. The entire leg can be visualized on this film from the hip to the ankle, or it can be adjusted to define the vascular tree to the bifurcation of the popliteal arteries (Fig. 2). The tube is centered over the tunnel. The tunnel used is pictured in Figure 3. It consists of two 1¼-in. plywood boards nailed together to form an oblong box, open on one side. On each end is a track which is set in the center of the tunnel. This track supports a lead-lined tray that is wide enough to hold one of the two 14×16 cassettes. In addition, one half of the length of the tunnel is lead-covered, to protect the top cassette in the tray. On the unprotected portion of the tunnel is placed a Lysholm grid.

The first cassette is placed on the bottom of the tunnel in the section that is not covered by the lead shield. The second cassette is placed on the lead-lined tray which is positioned under the protective lead shield. Upon completion of the third injection, an x-ray exposure is taken, the tray with the upper casette is pushed over to the unprotected portion of the tunnel with a short stick. A second x-ray exposure is then made. The first film is protected by the lead lining of the tray during this second exposure.

We have, in this manner, secured at least two films of any given area of the vascular system from the aorta to the midcalf or lower. By the foregoing technique of fractionation, we have eliminated the need of special apparatus and added an additional safety factor. The use of conventional 14×17 films eliminates the difficulty of storing large 14×36 films. Injections of large amounts of a radiopaque medium in the periaortic region are

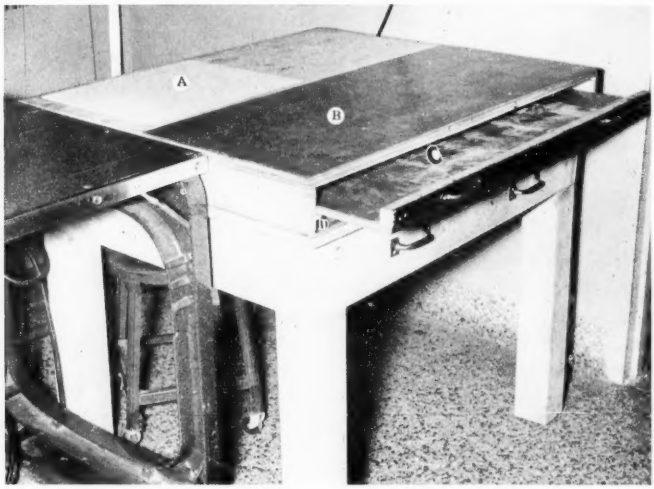

Fig. 3.—*A*, Lysholm grid; *B*, lead covering over half of tunnel; *C*, lead-lined tray which holds the second cassette, and *D*, opening through which first cassette is slipped to the unprotected portion of tunnel.

also eliminated. Poor-risk patients are not subjected to massive injections of radiopaque medium which they may not tolerate.

Aside from the additions of a wooden cassette tunnel, two 14×36 cassettes, and a Lysholm grid, a conventional tube stand of at least 52 in. is also required. No large Bucky equipment is needed. Since the injection can be done automatically, protection of the operator against stray radiation is complete.

SUMMARY

A new technique of introducing a radiopaque medium by fractionation in translumbar aortography is presented.

It is a safe and simple method which will help prevent serious sensitivity reactions.

The entire vascular tree is visualized from the aorta, in the region of the renal arteries, to the dorsal pedis vessels.

No expensive additional equipment is required.

The operator is protected against excessive stray radiation.

Stanley Schulman and Malcolm Haber, our technical staff, cooperated in this process.

1903 Avenue K (30).

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Rokitansky-Aschoff Sinuses as Related to Chronic Cholecystitis

LEONARD K. STALKER, M.D.
BAXTER DAVIES, M.D.
and
T. PAUL GUEST, M.D., Rochester, N. Y.

The so-called Rokitansky-Aschoff sinuses of the gall bladder are very rarely visualized roentgenographically. Less than a dozen proved cases have been reported in the literature. When radiographic visualization of these sinuses does occur, a definite diagnosis of chronic cholecystitis can be made and surgery is indicated. The pathologist frequently sees what are called Rokitansky-Aschoff sinuses, but it is rare for the surgeon to grossly identify these, and even rarer for the roentgenologist to visualize them.

The term Rokitansky-Aschoff sinuses, or Luschka's crypts of the gall bladder, is applied to deep outpouchings of mucosa extending into or through the muscular coat and into the perimuscular layers, leading to separation of the interstices and delicate walls of the muscle bundles. Normally the gall bladder muscle bundles are resistant to such outpouchings. In the presence of inflammation, however, these contractile structures become weakened with obstruction and chronic overdistention and the mucosa can readily herniate through the intermuscular septa and extend into the perimuscular tissues, even reaching the serosa.

An excellent article was written on this pathological condition by Robertson and Ferguson. These authors examined 495 gall bladders, 175 postmortem specimens and 320 from surgical operations. They noted that there was an increase in both the number and the depth of Luschka's crypts (Rokitansky-Aschoff sinuses) in gall bladders in which chronic inflammation has been present. They described these sinuses as the indentations of the mucosal epithelium which range from shallow grooves or pouches, which hardly merit the qualifying word pathologic, to deeply penetrating, complexly branching invaginations. They noted that when these structures became partially or completely cut off from the surface they became dilated in whole or in part and often assumed the characteristics of the so-called adenoma. On occasion, bile pigments and irregularly arranged collections of cholesterol crystals were observed in the crypts, but, as a rule, these contained a transparent mucin-like fluid. All degrees of inflammation were seen, including abscess formation, complete obliteration of the epithelial cells that normally line the space, and even rupture into the peritoneal cavity or the liver. Generally, anatomic signs of inflammation were absent, and there is little reason to conclude that infection or irritation played other then a secondary role.

It was the feeling of these authors that the initial invagination of the mucosa of the gall bladder in underlying layers was unquestionably produced by increased intracystic pressure, perhaps with relaxation of the elements which composed the wall. They felt that the further development of the diverticula, particularly budding of pouches and cyst formation, may be aided by the pressure of contained secretions. In their series approximately half of all gall bladders removed from persons more than 30 years of age showed invagination (Rokitansky-Aschoff sinuses) of the mucosa into the underlying structures.

It was the feeling of Robertson and Ferguson that this situation was caused by increased intracystic pressure, the result of neurogenic dysfunctional states of the extrahepatic biliary system, but in some cases mechanical factors such as stones and inflammatory obstructions were considered contributory. They felt that these crypts corresponded in every respect to the so-called diverticula of the colon and urinary bladder and should be denominated "diverticula of the gallbladder." They were described by Rokitansky and Aschoff and at the same time by Luschka and others. For this reason it was felt that there was little except custom to justify calling them Luschka's crypts or Rokitansky-Ascoff sinuses. In all probability the term diverticula of the gall bladder would be preferable.

The first radiological referrence to the Rokitansky-Aschoff sinuses was made by McCorkle and Fong in their description of a case of acute pneumocholecystitis in 1942. Subsequently, Ackerman described a case of diverticulosis of the gall bladder in 1948. It remained for Bean and Kocher, in 1950, to bring this condition forth as an entity in American radiologic literature. They described the gall bladder as showing moderate concentration of the dye, with numerous round-bottom, flash-like outpouchings extending from the mucosa into a thickened gall bladder wall. No shrinkage of the gall bladder was noted after a fatty meal. They felt that a stricture was present in the proximal portion of the gall bladder and that the Rokitansky-Aschoff sinuses were found distal to this. Some authors in reviewing this subject have thought that this stricture was congenital in nature, but others think that it is due to macroscopic sinuses containing pigment.

We recently treated a patient who presented a classic picture of chronic cholecystitis and in whom Rokitansky-Aschoff sinuses were demonstrated preoperatively and a positive diagnosis of chronic cholecystitis was made postoperatively. We thought that a detailed report of this case would be of interest.

The patient, a 39-year-old white woman, was seen in May, 1954. She complained of recurrent attacks of acute indigestion and a persistent chronic gaseous dyspepsia of 18 months' duration. These attacks had progressively increased in frequency so that during the past six months they had occurred twice weekly. They were usually initiated by the ingestion of fatty foods and were manifested by epigastric fullness, "heart-burn," pain between the shoulder blades, nausea, and frequently vomiting. She had consulted several physicans, and when two radiographic studies of the gall bladder, together with a complete gastrointestinal series, were reported negative her symptoms were attributed to nerves. She was subsequently seen by us. A complete physical examination with examination of the blood and urine revealed nothing of significance. We were so impressed with the history of biliary tract disease that repeated roentgenograms were suggested.

Fig. 1.—Roentgenogram showing Rokitansky-Aschoff densities.

The gall bladder was examined radiographically by the usual technique and revealed excellent dye concentration. General characteristics appeared anatomical. However, a distinct rosary of small oval densities was demonstrated surrounding the contour of the gall bladder. The texture of these unusual beads appeared similar to the opaque

material within the gall bladder itself. All these small bodies were closely related but showed no visible connections with the dye-filled fundus. There was minimal contraction of the neck. The important and significant finding of the opaque beads was best studied on the postprandial films. No translucent or opaque stones were visualized and no intramural calcifications were demonstrated.

The impression from the x-ray examination was that the chain of densities noted about the periphery of the gall bladder represented Rokitansky-Aschoff sinuses (Fig. 1).

At operation the gall bladder was found to be chronically diseased. There were a few surrounding adhesions. The wall was at least 1.5 cm. in thickness. On palpation the gall bladder was compressible and no stones were felt. Running through the wall one could feel small 0.5- to 1-cm.-sized cystic-feeling nodules. This gave one the impression almost that there were multiple small polyps attached to the wall of the gall bladder. No inflammatory obstruction was present. The liver, pancreas, and biliary ducts were grossly normal. The bed of the gall bladder was more vascular than normal, but a cholecystectomy was performed without event.

Pathological examination confirmed the gross findings. The mucosal lining was unremarkable. Scattered round cells were seen in the submucosa, which was thickened and edematous, as were the muscular coats. Markedly dilated mucosal glands containing amorphous matter were seen within the wall. Some of them contained dense, dark brown amorphous material, presumably bile precipitate. A pathological diagnosis of chronic cholecystitis with Rokitansky-Aschoff sinuses was made (Fig. 2).

The patient had an uneventful convalescence and was dismissed on her 11th postoperative day. She has been carefully followed for a period of six months; her digestive symptoms have completely disappeared, and she has gained 15 lb. in weight.

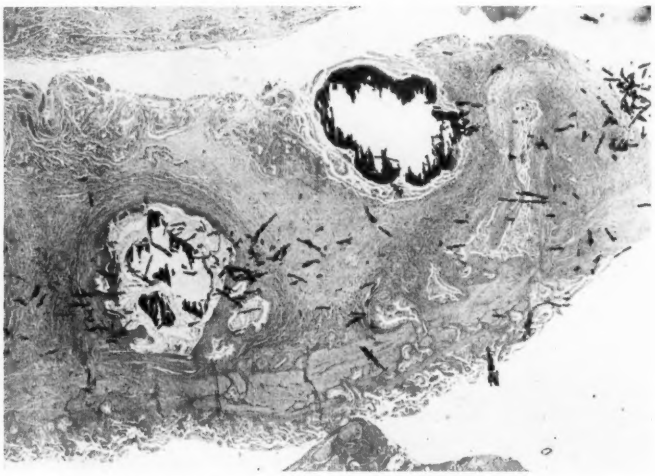

Fig. 2.—Photomicrograph showing Rokitansky-Aschoff sinuses.

COMMENT

This is an interesting case that has illustrated the occasional role of chronic cholecystitis in the presence of Rokitansky-Aschoff sinuses. The results obtained in the treatment of this case, together with the opinion in the literature, would indicate that whenever such a condition can be diagnosed roentgenographically surgery is indicated. We are of the opinion that a few patients have not received the desirable therapy for this problem, largely owing to the fact that the roentgenologist could not demonstrate stones or other specific disease.

Rokitansky-Aschoff sinuses are not found more frequently roentgenographically because of many factors. Complete failure of dye concentration from disease with nonvisualization is presumably the most frequent reason. Size of the sinus orifices, inspissated bile, and related cellular deposits may certainly prevent the opaque medium from entering the sinuses. Nonuse of the fatty meal

with associated delayed film also could possibly result in poor visualization. There are possibly other reasons, such as misinterpretation and poor film technique, but these factors can easily be eliminated. Realization of these various errors should stimulate our diagnostic acumen and result in more complete and thorough examination.

SUMMARY

A case of chronic cholecystitis in which preoperative visualization of Rokitansky-Aschoff sinuses was obtained is presented.

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SURGERY ILLUSTRATED

Pulmonary Hamartoma, an Enlarging Neoplasm

WILSON WEISEL, M.D
MARVIN GLICKLICH, M.D.
and
FRANCIS B. LANDIS, M.D., Milwaukee

Among the benign thoracic neoplasms, pulmonary hamartomas have been considered one of the most innocent. The 10 patients whose cases are reported in this paper had lesions of this type which we have considered worthy of recording because all of the tumors showed varying degrees of enlargement on roentgen examination. The tumors were considered possible carcinoma in nine instances and tuberculoma in one before a definite pathologic diagnosis was made.

REPORT OF CASES

CASE 1.—S. M. (VAH 151921), a 38-year-old white man, was admitted to the hospital, Sept. 6, 1950, because of a round lesion of the left lung which had increased 1.0 cm. in diameter during the preceding year. The patient had no pulmonary symptoms, and physical examination revealed no abnormalities. Skin tests to PPD, blastomycin, coccidioidin, and histoplasmin were negative. Bronchoscopy was not contributory. Roentgenograms of the thorax (Fig. 1) revealed a round lesion, 1.5 cm. in diameter, in the superior segment of the left lower lobe. Review of the patient's previous film showed that the lesion had increased 1.0 cm. in diameter during the preceding year.

Thoracotomy and superior segmentectomy were performed on Sept. 16, 1950, with the use of a posterolateral approach. The coin-type tumor proved to be a well-circumscribed hamartoma. Histologically, the tumor consisted of cartilaginous islands and clefts of columnar epithelium in a fibrous capsule. The postoperative course was uneventful.

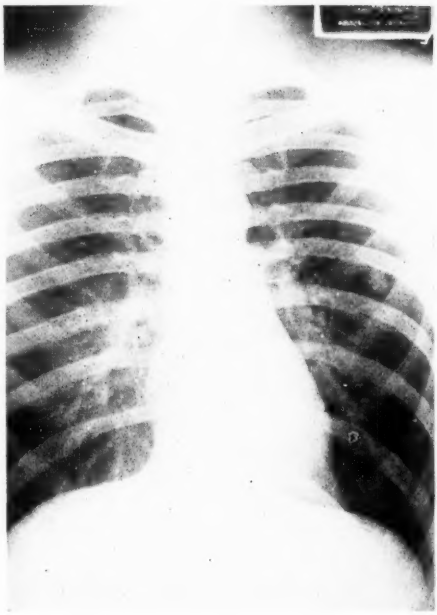


Fig. 1.—Anteroposterior thoracic roentgenogram, showing 1.5-cm. round lesion in left midlung field between posterior 7th and 8th ribs.

CASE 2.—L. X. P. (VAH 137014), a 32-year-old white man, was admitted to the hospital, June 1, 1951, because of a round lesion of the right lung. He was asymptomatic and the only abnormal physical findings were decreased breath sounds over the right lower chest in the midaxillary line. Thoracic roentgenograms (Fig. 2) demonstrated a round mass 5.0 cm. in diameter in the right lower lung field. A survey chest x-ray taken in 1949 was reviewed, and the same lesion was apparently present in the right lower lung field although it seemed to be just one-half the size at the time of hospital admission. At bronchoscopy, a globular mass appeared to obstruct the right medial basilar bronchus, but bi-

From the Thoracic Surgical and Medical Services of the Veterans Administration Center, Wood, Wis.; St. Joseph's Hospital, and the Marquette University School of Medicine.

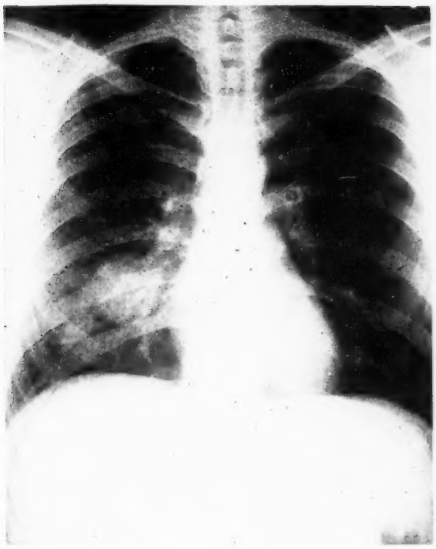
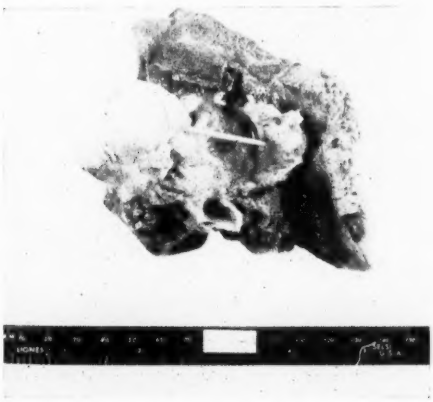


Fig. 2.—Anteroposterior and right lateral x-rays of chest with 5.0-cm. round lesion in right lower lung field.

opsy demonstrated only cartilage and bronchial mucosa.

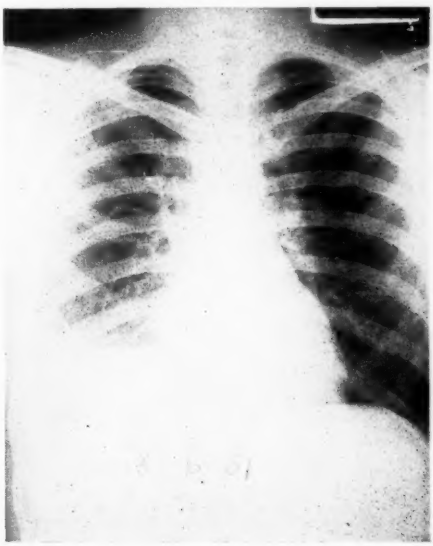
Right thoracotomy was performed on June 6, 1951, with the use of a posterolateral, sixth interspace approach to the pleural space. A right lower lobectomy was performed. A yellow-gray mass 6.0 cm. in diameter occupied the center of the lobe. The lesion was attached to the medial basilar bronchus by a pedicle, measuring 1.5 cm. across its base, and protruded into the bronchus (Fig. 3). Histologically, the tumor was made up of cartilaginous islands, clefts of bronchial epithelium, mucus glands, fat, lymphocytes, and myxomatous tissue. The patient's postoperative course was uneventful (Fig. 4).

Fig. 3.—Gross specimen (Case 2), showing right lower lobe with 6.0 cm. hamartoma attached by stalk to the medial basal bronchus.



Case 3.—F. R. (VAH 144412), a 58-year-old white man, was admitted Aug. 28, 1951, because of

Fig. 4.—Postoperative roentgenogram (Case 2) after right lower lobectomy.



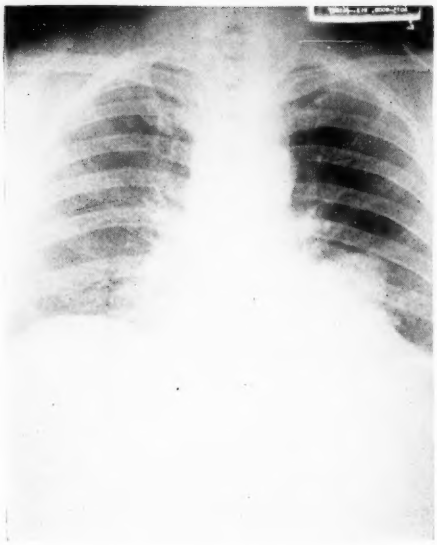
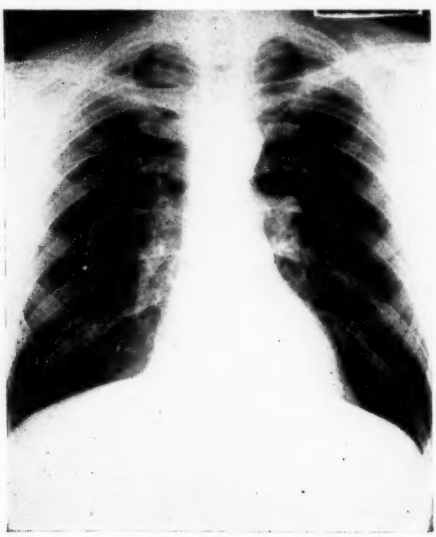


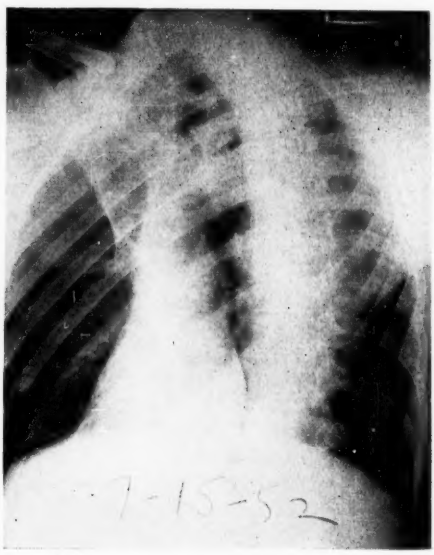
Fig. 5.—Anteroposterior and left lateral thoracic roentgenograms showing large mass in lingular segment of left upper lobe.

a round lesion of the left lung which had more than doubled in size over a period of two years. The patient had no symptoms referable to the respiratory tract, and physical examination had not been contributory. Thoracic roentgenograms (Fig. 5) showed a rounded density in the anterior area of the left lower lobe. Bronchoscopy findings suggested an extrabronchial lesion of the left upper lobe.

On Oct. 9, 1951, a left thoracotomy was performed through the periosteal bed of the resected fifth rib, with the use of a posterolateral approach. A large mass in the lingula of the left upper lobe was removed by a subsegmental resection. The

Fig. 6.—Anteroposterior and left anterior oblique chest x-rays showing 1.5 cm. round lesions in middle lobe of right lung.





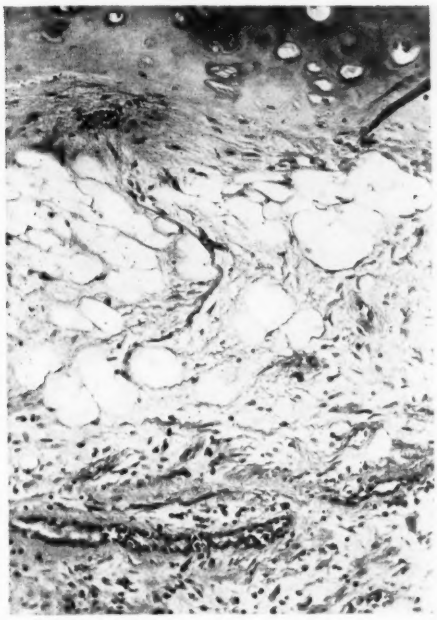


Fig. 7.—Microscopic section of hamartoma (Case 5) showing cartilage, myxomatous tissue, and epithelium-lined cleft; reduced to ⅕ of mag. × 65.

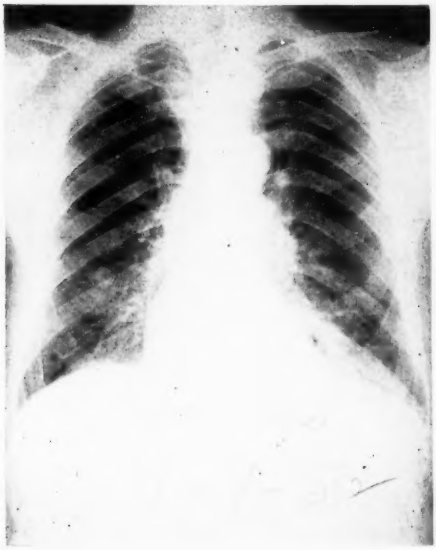
Fig. 8.—Anteroposterior and left anterior oblique chest x-rays demenstrating hamartoma in right middle lobe.

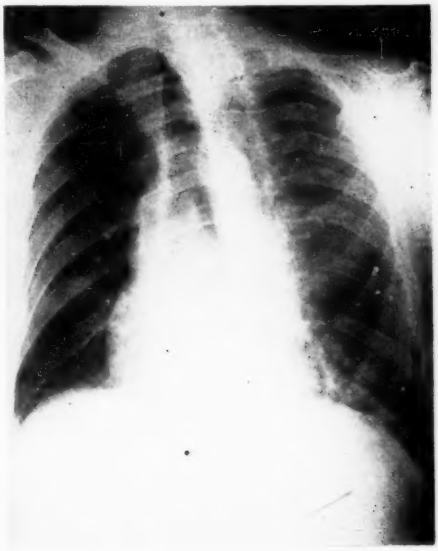
lesion proved to be a lobulated yellow-gray tumor measuring 10 cm. in its greatest diameter, with a 1.5-cm. central cystic area containing mucinous material. Histologically, the tumor was made up of a fibrous capsule, columns of cartilage, and masses of myxomatous cells. The patient's postoperative course was satisfactory.

Case 4.—H. E. E. (VAH 140297), a 46-year-old white man, was admitted to the hospital July 14, 1952, because of a round lesion of the right lung which had increased in size 0.5 cm. over a six-month period. The patient was asymptomatic, and results of physical examination were within normal limits. A chest x-ray showed a round lesion of the lateral segment of the right middle lobe 1.5 cm. in diameter (Fig. 6).

At right thoracotomy on Sept. 2, 1952, a round lesion was removed from the lateral segment of the middle lobe by wedge resection. The nodule on cut section was 1.5 cm. in diameter and had a pearl-gray color. The bulk of the lesion, on microscopic examination, was made up of cartilaginous lobules, with interstitial areas of fibrous tissue, fat, and cuboidal epithelium. The patient's convalescence was uneventful.

Case 5.—J. M. M. (VAH 139562), a 65-year-old white man, was admitted to the hospital April 5, 1953, because of a round lesion in the anterior basal segment of the right lower lobe. It was thought that the lesion had increased a few millimeters in diameter over a one-year period. The lesion was removed by a subsegmental resection on April 9, 1953. On cut section the lesion was of cartilaginous consistency and pink-gray in color and measured





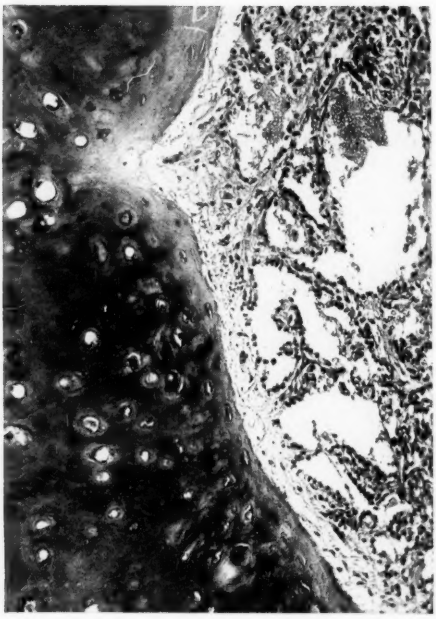
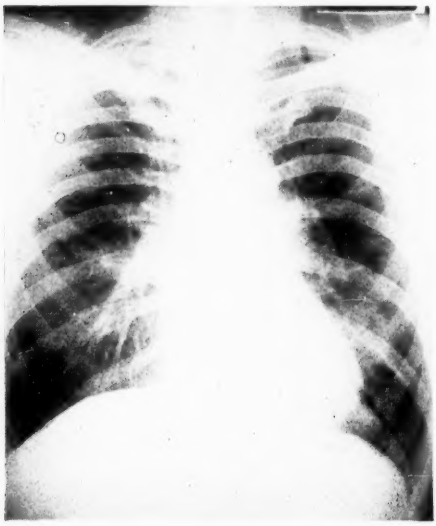


Fig. 9.—Microscopic sections (reduced to ⅕ of mag. × 150) of hamartoma showing cartilage and epithelium in a fibromyxomatous framework.

Fig. 10.—Anteroposterior x-ray of chest showing round lesion in right second anterior interspace and right apical cavitary and infiltrative lesion.



2.0 cm. in diameter. Histologically cartilage was the predominant tissue, with septa of fat, myxomatous tissue, and cuboidal epithelium (Fig. 7). The postoperative course of the patient was uneventful.

Case 6.—H. T. L. (VAH 141420), a 59-year-old white man, was admitted Oct. 9, 1952, because of a productive cough, dyspnea, and a round lesion of the right lung. A review of the patient's x-rays showed no evidence of the lesion in films taken five years previously and a lesion 1.5 cm. in diameter in the right lower lung as well as pneumonitis in the left lower lung (Fig. 8).

The round lesion was removed from the middle lobe by subsegmental resection at right thoracotomy on March 6, 1953, and proved to be a hamartoma on

Fig. 11.—Thoracic roentgenogram of Case 7, showing increase in size of nodule in right second interspace.

frozen-section examination. Microscopy demonstrated a fibrous capsule surrounding islands of mature cartilage, with fibrous tissue and bronchial epithelium between the islands (Fig. 9). The patient's convalescence was uneventful.

Case 7.—W. B. (VAH 145534), a 50-year-old white man, was hospitalized April 7, 1953, because a right apical lesion had been discovered on a routine mobile survey film two months previously. Thoracic roentgenograms (Fig. 10) revealed a dense infiltrate in most of the circle of the right 1st rib with a central area of rarefaction 2.0 cm. in diameter. In addition, a 1.0-cm. round nodule was present in the right second interspace. Sputum smears and cultures for acid-fast bacilli were positive.

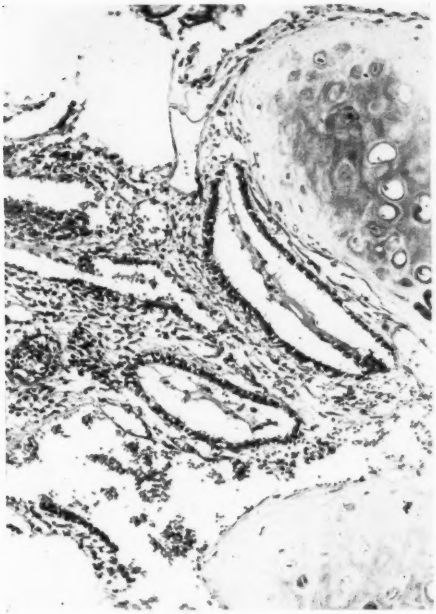


Fig. 12.—Histologic section of hamartoma (Case 7), showing cartilage, epithelium, and fibromyxomatous tissue (reduced to ⅓ of mag. × 100).

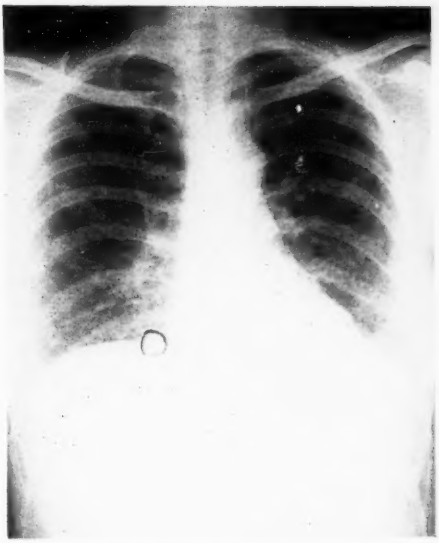
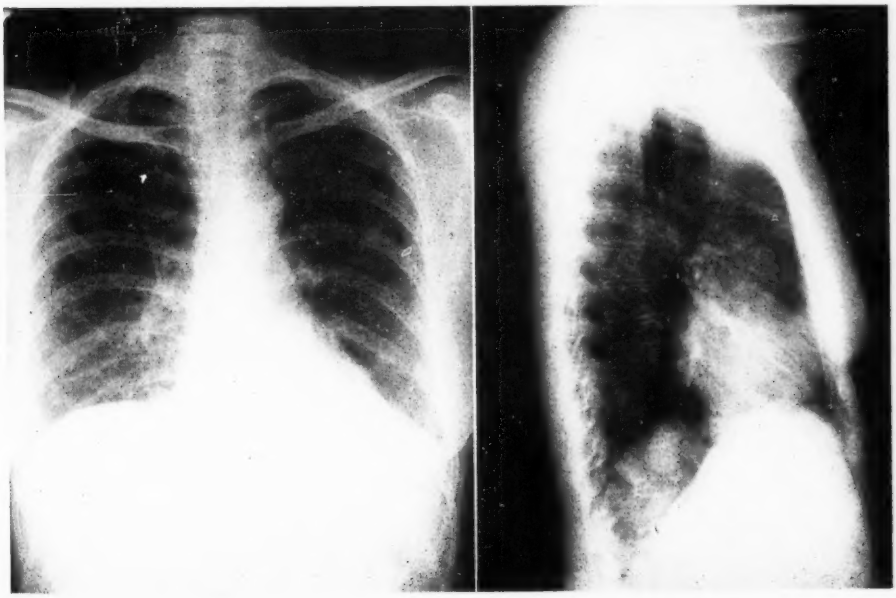


Fig. 13.—Chest x-ray showing round lesion of right lower lobe at cardiophrenic angle.

After 13 months' treatment with streptomycin and isoniazid, very little change had occurred in the roentgenograms. The patient's sputum remained positive; the apical cavity persisted, and the nodule

Fig. 14.—Anteroposterior and right lateral roentgenograms (Case 10), showing apparent increase in size of round lesion in right lower lobe compared to Figure 13.



in the right second interspace had increased slightly in size (Fig. 11). A resection of the right apical and posterior segments of the upper lobe was performed on June 10, 1954, with a posterolateral approach for the thoracotomy. The nodular lesion proved to be a hamartoma 2.0 cm. in diameter which microscopically was composed of cartilaginous islands with irregular clefts lined by respiratory epithelium (Fig. 12). The cavitary lesion proved to be positive on smear and culture for tubercle bacilli. The patient's postoperative course was satisfactory.

CASE 8.—R. T. D. (VAH 134426), a 58-year-old white man, was admitted to the hospital on Sept. 22, 1951, and died as the result of uremia secondary to multiple myeloma on Nov. 20, 1951. Chest x-rays had revealed multiple osteolytic lesions of the rib and a round lesion in the right upper lung field, which had increased in size from 0.5 to 1.0 cm. diameter in one year's time.

At postmortem examination the nodule of the right upper lobe proved to be a gray-white lesion of cartilaginous consistency, about 2.0 cm. in diameter. Microscopic sections showed foci of cartilage surrounded by strands of loose myxomatous tissue, fat, nerve elements, and occasional strips of ciliated columnar epithelium.

CASE 9.—G. C. (VAH 139418), a 56-year-old white man, who was admitted to the hospital on June 2, 1952, died as the result of widespread metastases from an adenocarcinoma of the cecum and multiple pulmonary emboli on July 8, 1952. Although previous chest x-rays had been negative, his admission film showed a round lesion of the left lower lobe.

Fig. 15.—Microscopic section of hamartoma (Case 10), demonstrating masses of immature cartilage with clefts of epithelial cells (reduced to 1/7 of mag. × 150).

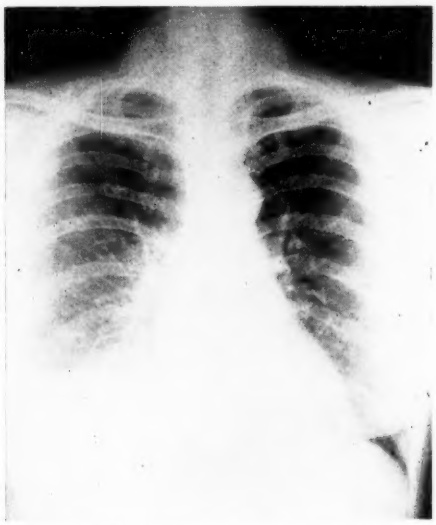


Fig. 16.—Postoperative chest x-ray (Case 10) after posterior basal segmentectomy.

At postmortem examination a round lesion was found in the left lower lobe, 2.0 cm. in diameter. Histologic examination demonstrated cartilaginous islands in a fibromyxomatous stroma, with intervening spaces lined by columnar epithelium.

CASE 10.—J. S. (St. J. 5056), a 50-year-old white housewife, was admitted to the hospital on April 23, 1954, because of a round lesion in the right lower lung field. The patient was asymptomatic, but chest x-rays taken three months previously had shown the lesion of the right lower lung. During this period of observation the round shadow had apparently increased 0.5 cm. in diameter (Figs. 13 and 14).

A right thoracotomy was performed on April 27, 1954. The pleural space was entered through a posterolateral incision, and the lung lesion was removed by a posterior, basal segmental resection. The tumor proved to be a 2.5 cm. white nodule which on cut section was seen to be of cartilaginous consistency. Microscopic sections showed a preponderance of cartilaginous tissue, with clefts of epithelium and fibromyxomatous tissue (Fig. 15). The patient's recovery was uneventful, and she was discharged on May 1, 1954 (Fig. 16).

COMMENT

Although the increasing interest in thoracic neoplasms has been vividly reflected in the rapidly accumulating accounts of pulmonary hamartomas,* the primary clinical in-

* References 2, 3, 6, 7, 10, and 15.

terest in these lesions has been in the difficult differential diagnosis the tumors present.† Considerable time elapsed after Albrecht [1] coined the classification into which these tumors might be included and the recognition of pulmonary types of the neoplasm, but by 1950 [3] more than 100 hamartomas of the lung had been recorded. Owing to the work of Womack and Graham,[16] the dangerous potential in relatively benign lesions of the lung have been recognized, and a few reports have pointed out the clinical hazards or pathologic instability of some hamartomas‡

The problems of diagnosis are well illustrated by the cases presented and a detailed review of the patients has not been clarifying, for in only one of the patients (Case 3) was calcification noted in the roentgenogram shadow, but calcium was found histologically in two (Cases 3 and 9). Six of the lesions were found in the right lung and four in the left, but they were evenly distributed in the various lobes and in depth of residence within the lung.

The lesions studied in this group have been a selected series, but pathologically (except for Case 3), the tumors have not presented unusual histologic features. In none of the patients have lymph node metastases been demonstrated, or have multiple tumors or recurrent tumors been encountered.

SUMMARY

Ten patients are presented who had pulmonary hamartomas which showed growth or enlargement of varying degree on roentgen examination.

† References 8, 9, and 12.

‡ References 4, 5, 11, 13, and 14.

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Scientific Exhibits

THE FUNCTION OF THE GASTRIC ANTRUM IN HEALTH AND DISEASE

LESTER R. DRAGSTEDT, M.D., Ph.D.; EDWARD R. WOODWARD, M.D.
HARRY A. OBERHELMAN Jr., M.D.; SHIRL O. EVANS, M.D.
STANLEY P. RIGLER, M.D.; JOHN H. LANDOR, M.D.
LESTER R. DRAGSTEDT II, M.D., and
EDWARD S. LYON, M.D., Chicago

EXPERIMENTAL proof has been secured supporting the postulate of Edkins that the gastric phase of secretion is mediated by the antrum of the stomach, which functions as an endocrine organ, releasing the gastric secretory stimulant, gastrin, under appropriate conditions. As is indicated in Figure 1, the release of gastrin from the mucous membrane of the antrum is brought about by local stimulation, by food, peristalsis, and distention. Inhibition of antrum function results from the contact of acid with the antrum mucosa when the concentration of acid reaches a critical level.

Experimental proof that gastrin is liberated from the antrum on contact with food or as a result of distention is illustrated in Figure 2. Food plus distention is a more adequate stimulus for gastrin release than either distention or food alone. A hypersecretion of gastric juice of antrum origin may be induced by transplanting the antrum of the stomach into the colon as a diverticulum. This hypersecretion is abolished if the colon containing the diverticulum is excluded from gastrointestinal continuity and washed free of fecal material or food. Obstruction to the isolated segment of colon containing the antrum transplant produces a vigorous secretion of gastric juice, even though no food or other material is introduced into the colon. This stimulation of the transplanted antrum is brought about by the excessive peristalsis in the obstructed, isolated colon.

Proof for the statement that acid applied to the antrum mucosa inhibits the gastric phase of secretion is provided by experiments in Figure 3. The application of acid solutions to the antrum mucosa, however, failed to abolish the secretion of gastric juice induced by insulin hypoglycemia or by the presence of food in the intestinal tract. These findings suggest that the application of acid abolishes the antrum phase of secretion by a local effect and not through the liberation of an inhibitory chemical substance into the blood stream. The application of cocaine to the antrum mucosa prevents the release of gastrin by the application of either food or distention.

Hyperfunction of the gastric antrum resulting in hypersecretion can be produced by increasing those factors which stimulate the antrum, decreasing those factors which inhibit the antrum, or a combination of the two factors. The net result is to cause the release of an excessive amount of gastrin or a prolongation of the period of

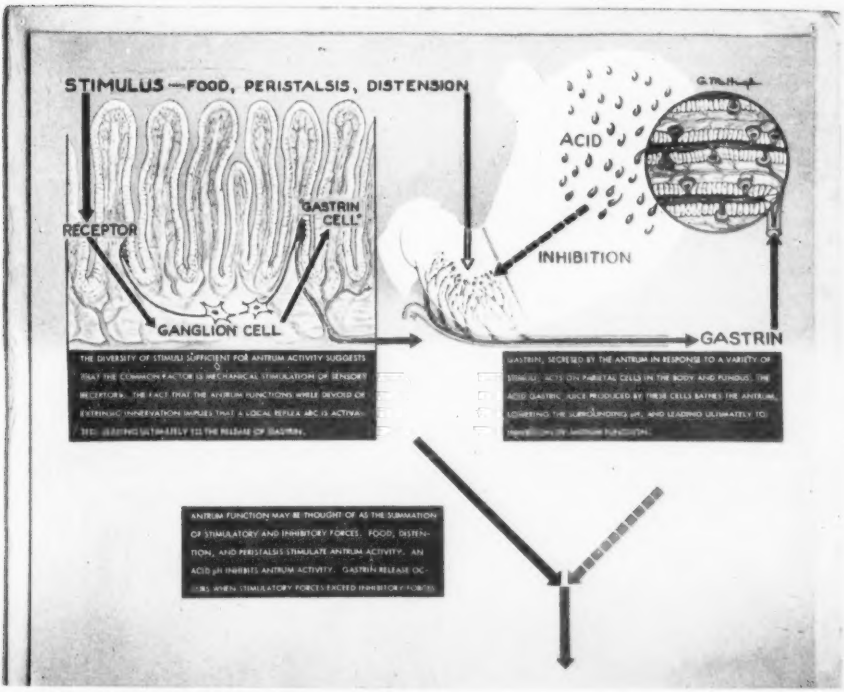
From the Department of Surgery of The University of Chicago.

Shown as a scientific exhibit of the Section on Surgery, General and Abdominal at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 21-25, 1954.

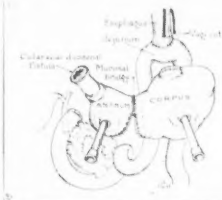
THE FUNCTION OF THE GASTRIC ANTRUM IN HEALTH AND DISEASE

The Gastric Phase of Secretion

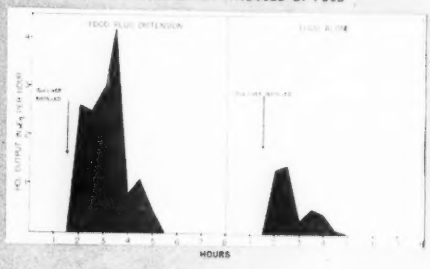
IS MEDIATED BY GASTRIN, A HORMONE LIBERATED BY THE ANTRUM IN RESPONSE TO LOCAL STIMULATION


STIMULUS—FOOD, PERISTALSIS, DISTENSION
RECEPTOR
GASTRIN CELL
GANGLION CELL
ACID
INHIBITION
GASTRIN
THE DIVERSITY OF STIMULI SUFFICIENT FOR ANTRUM ACTIVITY SUGGESTS
ANTRUM FUNCTION MAY BE THOUGHT OF AS THE SUMMATION
OF STIMULATORY AND INHIBITORY FORCES. FOOD, DISTEN-
TION, AND PERISTALSIS STIMULATE ANTRUM ACTIVITY. AN
ACID pH INHIBITS ANTRUM ACTIVITY. GASTRIN RELEASE OC-
CURS WHEN STIMULATORY FORCES EXCEED INHIBITORY FORCES

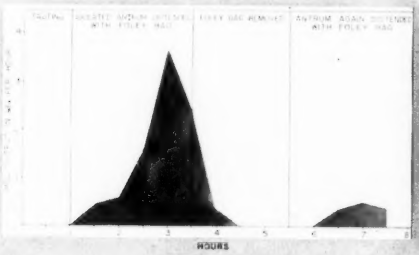
STIMULATING FACTORS



ANTRUM STIMULATION PRODUCED BY FOOD



ANTRUM STIMULATION PRODUCED BY DISTENTION



THE EFFECT OF STIMULATION OF ISOLATED ANTRUM POUCHES ON GASTRIC SECRETION

PERFUSION OF AN ISOLATED ANTRUM POUCH WITH A VARIETY OF SUBSTANCES STIMULATES SECRETION

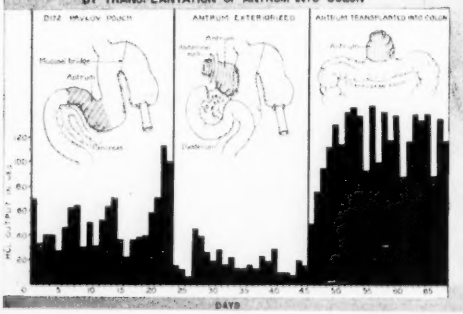
SIMPLE DISTENSION OF THE POUCH STIMULATES SECRETION

ACID					
TAP WATER					
MILK					
BILE					
PANCREATIC JUICE					
FEEDS					
DISTENTION					
AMINOSOL					
AMYNOSOL					
FRESH LIVER					
FRESH LIVER					
FRESH LIVER					
FRESH LIVER					
FRESH LIVER					
RAW MEAT					

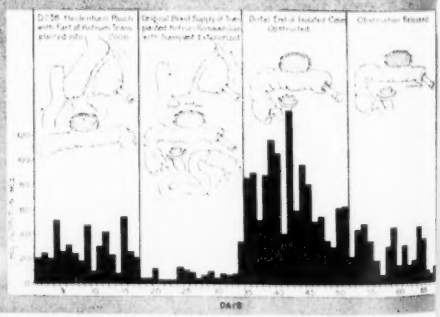
*ANIMAL RETCHED

TRANSPLANTATION OF THE ANTRUM TO THE COLON PRODUCES A HYPERSECRETION OF GASTRIC JUICE. DISTENSION AND PERISTALSIS ARE PROBABLY THE MAJOR STIMULI.

HYPERSECRETION IN PAVLOV POUCH PRODUCED BY TRANSPLANTATION OF ANTRUM INTO COLON

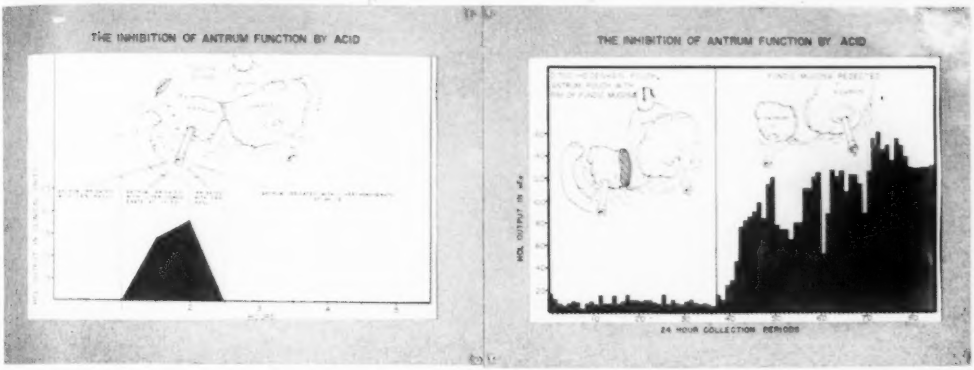


THE EFFECT OF DISTENTION ON THE DENERVATED TRANPLANTED ANTRUM

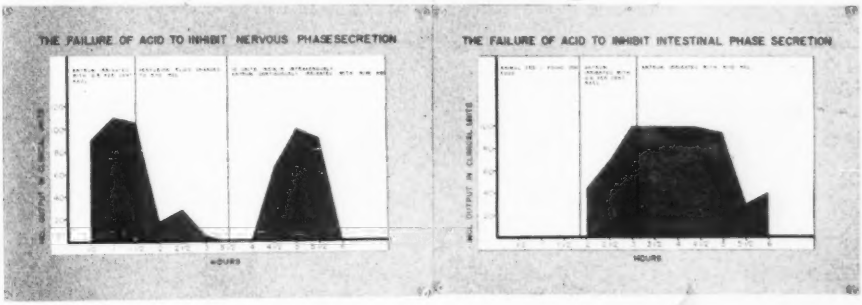


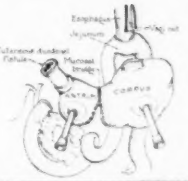
INHIBITING FACTORS

STIMULI USUALLY SUFFICIENT FOR ANTRUM FUNCTION ARE RENDERED INEFFECTIVE BY AN ACID MEDIUM

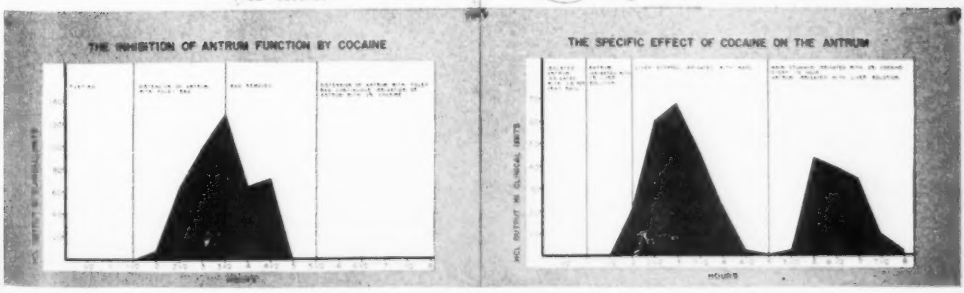


THE INHIBITING EFFECT OF ACID DOES NOT ALTER NERVOUS AND INTESTINAL PHASE SECRETION. IT SPECIFICALLY INHIBITS THE RELEASE OF GASTRIN


THE FAILURE OF ACID TO INHIBIT NERVOUS PHASE SECRETION
THE FAILURE OF ACID TO INHIBIT INTESTINAL PHASE SECRETION
HOURS
HOURS



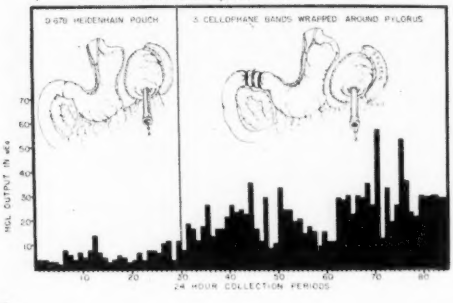
TOPICAL APPLICATION OF COCAINE TO THE ANTRUM MUCOSA PREVENTS THE STIMULATING EFFECT OF DISTENSION. THE SENSORY LIMB OF THE REFLEX ARC IS PROBABLY BLOCKED.



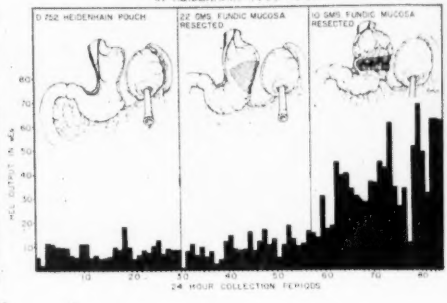
HYPERFUNCTION OF THE GASTRIC ANTRUM CAN BE PRODUCED BY INCREASING THE STIMULATORY FACTORS, DECREASING THE INHIBITORY FACTORS, OR A COMBINATION OF BOTH. THE NET EFFECT IS THE RELEASE OF AN EXCESS AMOUNT OF GASTRIN.

DELAYED EMPTYING OF THE STOMACH LEADS TO PROLONGED ANTRAL STIMULATION, AND A HYPERSECRETION OF ANTRAL ORIGIN

EFFECT OF PYLORIC OBSTRUCTION ON GASTRIC SECRETION IN HEIDENHAIN POUCH DOG

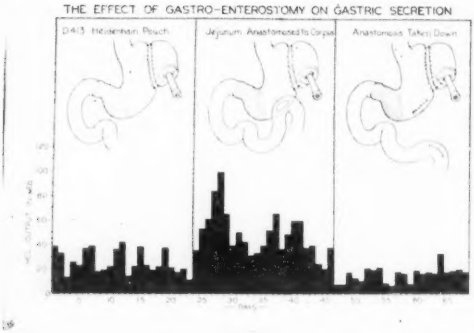


EFFECT OF PARTIAL FUNDUSECTOMY ON GASTRIC SECRETION IN HEIDENHAIN POUCH DOG

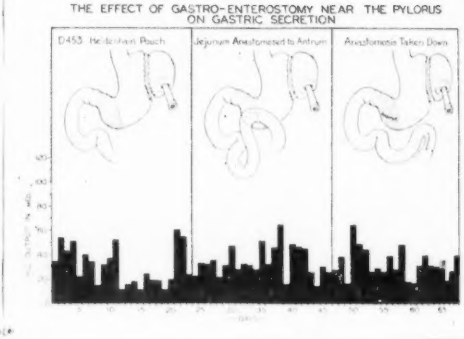

D 752 HEIDENHAIN POUCH
22 GMS. FUNDIC MUCOSA RESECTED
10 GMS. FUNDIC MUCOSA RESECTED
HCL OUTPUT IN mEq
24 HOUR COLLECTION PERIODS

FUNDIC GASTRO-ENTEROSTOMY DRAINS AWAY MUCH OF THE ACID PRODUCED BY THE PARIETAL CELLS. DECREASED ACID INHIBITION OF THE ANTRUM RESULTS

THE EFFECT OF GASTRO-ENTEROSTOMY ON GASTRIC SECRETION

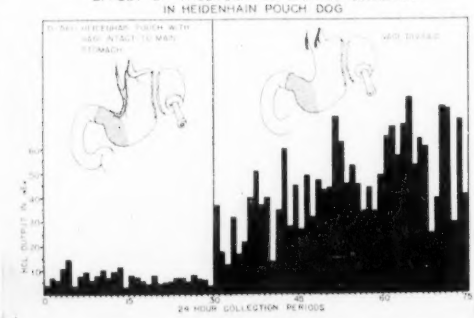


THE EFFECT OF GASTRO-ENTEROSTOMY NEAR THE PYLORUS ON GASTRIC SECRETION

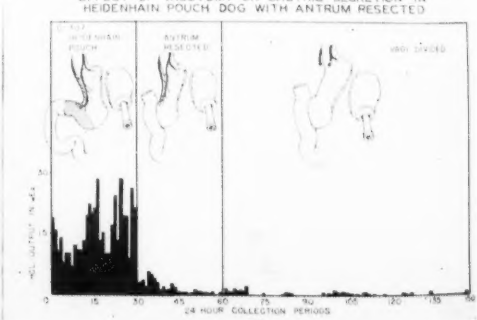


VAGOTOMY ELIMINATES THE CEPHALIC PHASE OF SECRETION, AND ALSO INCREASES THE EMPTYING TIME OF THE STOMACH. PROLONGED ANTRAL STIMULATION AND ANTRAL HYPERFUNCTION FOLLOW

EFFECT OF VAGOTOMY ON GASTRIC SECRETION IN HEIDENHAIN POUCH DOG



EFFECT OF VAGOTOMY ON GASTRIC SECRETION IN HEIDENHAIN POUCH DOG WITH ANTRUM RESECTED



CLINICAL IMPLICATIONS

GASTRIC AND DUODENAL ULCERS ····· PEPTIC ULCERS WITH DIFFERENT ETIOLOGIES

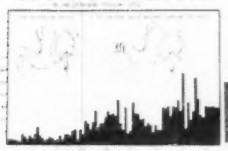
PATIENTS WITH DUODENAL ULCER HAVE NOCTURNAL SECRETIONS WELL ABOVE NORMAL LIMITS, WHILE PATIENTS WITH GASTRIC ULCER HAVE LOW OR NORMAL FASTING SECRETIONS. VAGOTOMY REDUCES THE HYPERSECRETION OF DUODENAL ULCER PATIENTS AND PERMITS THE ULCER TO HEAL. VAGOTOMY FOR GASTRIC ULCER FREQUENTLY LEADS TO RECURRENCE.

ANTRUM RESECTION FOR DUODENAL ULCER IS FOLLOWED BY A HIGH PERCENTAGE OF RECURRENT STOMAL ULCERS. ANTRUM RESECTION HEALS GASTRIC ULCERS AND RARELY RESULTS IN RECURRENT STOMAL ULCERS.

TWELVE HOUR NIGHT SECRETION

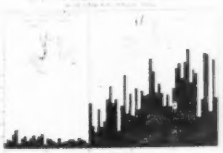
	NO OF PATIENTS	VOLUME IN CC	FREE ACID CL UNITS	HCL OUTPUT mEq
NORMAL	32	562	30	186
DUOD ULCER	309	1085	52	63
VAGOTOMY	309	521	10	5
GASTRIC ULCER	23	615	19	12

EXPERIMENTAL FACT ·· ·· CLINICAL CORRELATE



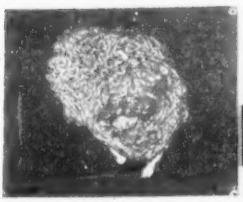
PARTIAL PYLORIC OBSTRUCTION LEADS TO DELAYED EMPTYING, PROLONGED CONTACT OF FOOD WITH THE ANTRUM, AND A HYPERSECRETION OF ANTRAL ORIGIN

MANY PATIENTS WITH GASTRIC ULCER HAVE EVIDENCE OF STASIS AND DELAYED EMPTYING OF THE STOMACH.



VAGOTOMY ALONE LEADS TO STASIS, PROLONGED ANTRAL STIMULATION, AND A HYPERSECRETION OF ANTRAL ORIGIN

GASTRIC ULCERS HAVE OCCURED IN 7 OF 150 DUODENAL ULCER PATIENTS TREATED BY VAGOTOMY ALONE, BUT HAVE NOT FOLLOWED VAGOTOMY AND GASTRO-ENTEROSTOMY



IN THE DOG, EXPERIMENTAL ANTRUM HYPERFUNCTION LEADS TO FORMATION OF GASTRIC ULCERS

Experimental evidence and clinical observation suggest that while duodenal ulcer is a result of hypersecretion of nervous origin, gastric ulcer may be due to hyperfunction of the gastric antrum.

time during which gastrin is being released from the antrum mucosa. Experimentally, antrum hyperfunction with hypersecretion was produced in dogs by experimental pyloric obstruction (Fig. 4). Hyperfunction of the antrum was also induced by removing part of the acid-secreting body of the stomach so that acid inhibition of the antrum mucosa did not occur. Prolongation of antrum function with hypersecretion was also induced in dogs by dividing the vagus nerves to the stomach. It is probable that this factor of stasis in the stomach following vagotomy alone, with resultant hypersecretion of gastric juice of antrum origin, has been a factor in producing the unfavorable results seen in patients where vagotomy alone has been carried out in the treatment of duodenal ulcer.

The clinical applications of these experimental studies are briefly referred to in Figure 5. The evidence now seems impressive that duodenal ulcers are due to a hypersecretion of gastric juice of nervous origin whereas gastric ulcers are usually due to hypersecretion of gastric juice of humoral or hormonal origin and arise as a result of hyperfunction or prolonged function of the gastric antrum. These concepts help to explain some of the differences in the response of gastric and duodenal ulcers to surgical treatment.

RETROPERITONEAL TUMORS IN INFANTS AND CHILDREN

CHARLES A. KRUSE, M.D.
SANTA MONICA, CALIF.
WILLIAM H. SNYDER Jr., M.D.
E. M. GREANEY Jr., M.D.
AND
LAWRENCE CHAFFIN, M.D.
LOS ANGELES

THE CLINICAL, anatomical, and statistical features of an initial series of 88 retroperitoneal tumors in infants and children are depicted, together with the presenting symptoms, age incidence, and pathological types. Curability and the importance of early diagnosis is emphasized by recent reevaluation (to January, 1954) of 111 neuroblastoma and Wilms' tumor cases. The transabdominal surgical approach with initial ligation of the renal pedicle and inclusion of Gerota's fascia in the removal of a Wilms' tumor is shown to be essential for an adequate cancer operation.

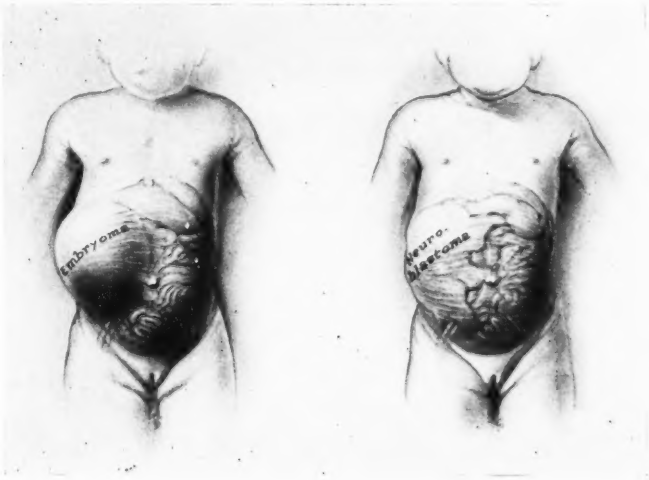
CLINICAL ASPECTS

Since retroperitoneal growths form the most frequent abdominal tumor of childhood, they are often encountered by the general surgeon. If one visualizes the growing retroperitoneal tumor as it pushes the posterior peritoneum forward and displaces

From the Surgical Service, Los Angeles Children's Hospital and the University of Southern California School of Medicine at Los Angeles.

Shown as a scientific exhibit of the Section on Surgery, General and Abdominal, at the 103rd Annual Meeting of the American Medical Association, San Francisco, June 21-25, 1954.

Fig. 1.—Artist's conception of developing retroperitoneal tumor, crowding the intra-abdominal organs.



the intra-abdominal organs, then one will understand the insidious nature of the onset and early similarity of symptoms of many of these tumors, regardless of whether they arise from the kidney or the sympathetic chain, or whether they are benign or malignant (Fig. 1).

In a previous report [1] we noted that the initial signs and symptoms in one-half of a group of retroperitoneal tumors were those of an enlarging abdomen and a palpable mass. In the other half, systemic manifestations were predominant. These were gastrointestinal disturbances, fever, pulmonary symptoms, anemia, and, occasionally, signs suggestive of an acute abdominal condition (Table 1).

TABLE 1.—*Presenting Symptoms of Retroperitoneal Tumors in Childhood*

Enlarging abdomen or mass	61%
Gastrointestinal	50%
Pain	30%
Constitutional	22%
Fever	16%
Genitourinary	12%
Trauma	6%

Clinical entities confused with retroperitoneal tumors in this series are listed in Table 2. Under the acute abdomen grouping, abdominal pain and tenderness were sufficiently impressive to result in appendectomy in four cases, and four others were observed for appendiceal abscess. A history of trauma with splinting of the left side suggested a diagnosis of ruptured spleen three times.

TABLE 2.—*Clinical Entities Confused with Retroperitoneal Tumors in Children's Hospital Series*

Blood dyscrasias	12
Acute abdomen	11
Splenomegaly	8
Tuberculosis	7
Mesenteric and dermoid cysts	7
Polycystic kidney	4
Orthopedic conditions	3

CLASSIFICATION

The following clinical classification of retroperitoneal tumors has been found to be generally acceptable: (*a*) embryoma of the kidney (Wilms' tumor), (*b*) neuroblastoma, (*c*) benign tumor, and (*d*) adrenal cortical adenoma. Table 3 indicates the relative frequency of occurence of the different types.

TABLE 3.—*Classification of Retroperitoneal Tumors* *

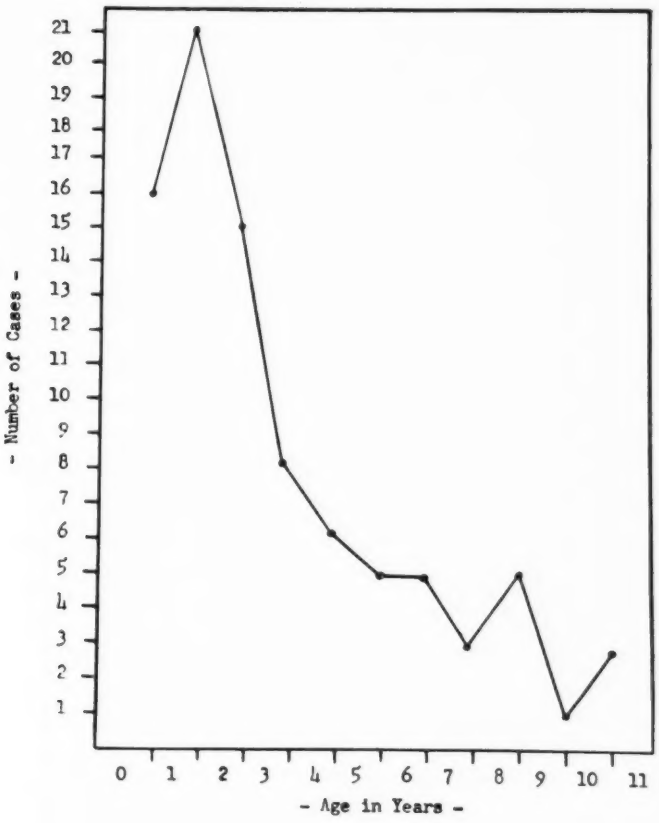
Series of 88 Consecutive Cases from Los Angeles Children's Hospital		
Type	Total Number	Percentage
Embryoma of kidney, or Wilms'	50	57
Neuroblastoma	34	39
Benign tumor	2	2
Adrenal cortical adenoma	2	2

* Pathological classification after operation or autopsy.

INCIDENCE AND SEX

Retroperitoneal tumors occurred, on the average, in 1 in 1000 patients consecutively admitted to Children's Hospital; however, they were responsible for 1 in 100 deaths. The average age of the patients was 38 months (Graph); 53 were boys and 35 were girls.

Graph I



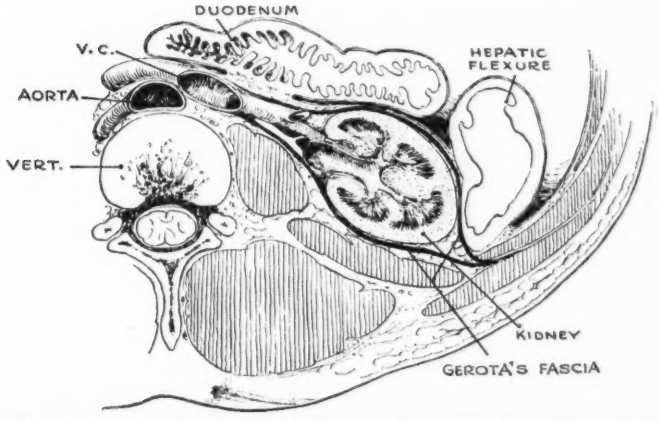


Fig. 2.—The dark line indicates Gerota's fascia, which surrounds the kidney. This is to be included in the resection of the retroperitoneal tumor.

ANATOMICAL FACTORS

Surgical Approach.—The transabdominal surgical approach with initial ligation of the renal pedicle and inclusion of both anterior and posterior layers of Gerota's fascia in the removal of a Wilms' tumor appears essential for an adequate cancer operation (Figs. 2 and 3).

Although no specific comparative data can be obtained from the literature and insufficient comparative statistics are available from our own series, it is apparent that the lumbar approach necessitates entering into the perirenal fatty tissue and incising

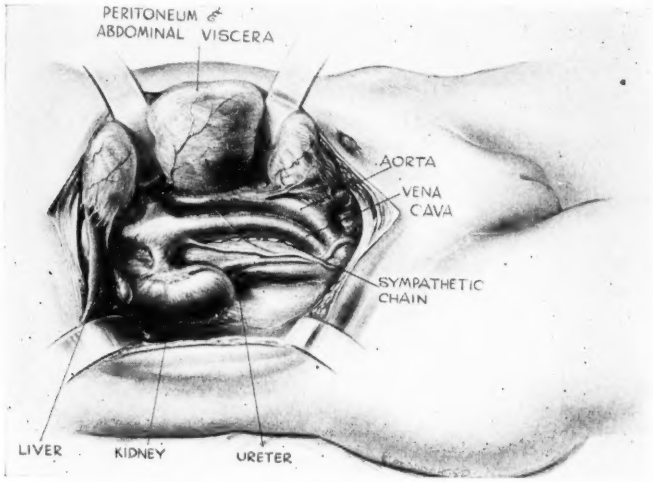


Fig. 3.—This indicates the approach through a right rectus abdominal incision. The retroperitoneal structures are exposed by retracing the ascending colon and its mesentery to the left side after the posterior peritoneum has been incised laterally. A transverse abdominal incision may be used as well, in most cases, and has certain physiological advantages.

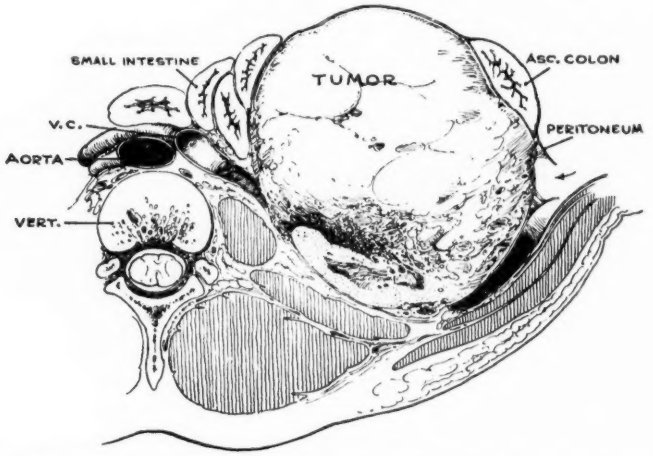


Fig. 4.—This is a cross-section showing the structures overlying the tumor in the retroperitoneal space.

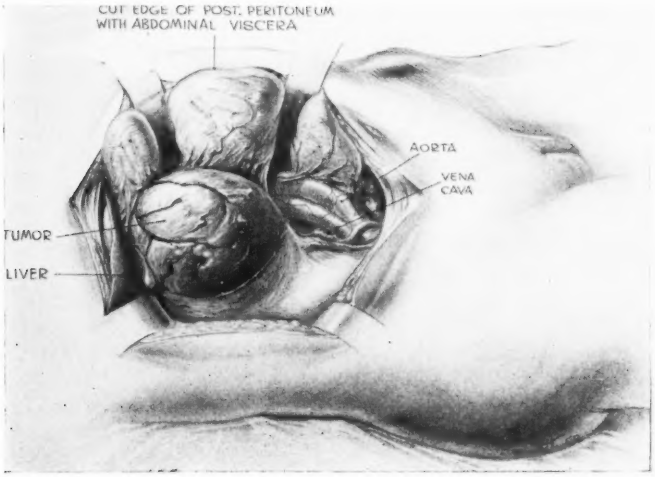
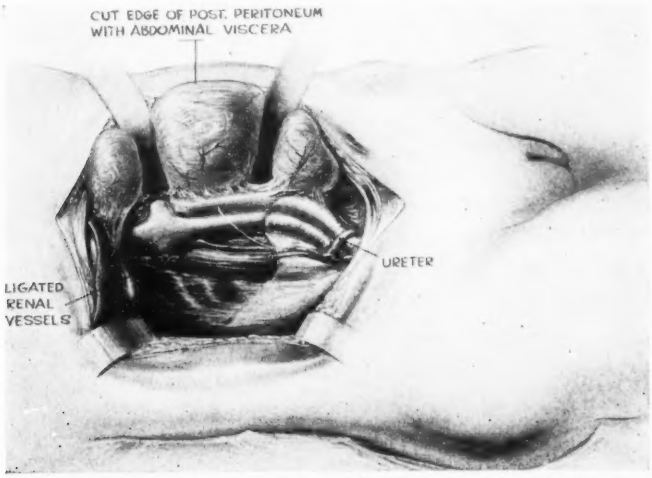


Fig. 5.—The posterior peritoneum has been severed laterally and the colon rotated to the left, exposing the tumor.

Gerota's fascia. Also the tumor is necessarily manipulated before ligation of the renal pedicle (Figs. 4 and 5).

A study of a significant series of renal neoplasms has been reported by McDonald and Priestly that revealed renal vein tumor thrombosis in the surgical specimens of 45% of the Wilms' tumor cases and 54% of the hypernephroma cases. A more recent report, 10 years later, from the same institution on collateral venous drainage from the kidney produced by occlusion of the renal vein in the experimental animal suggests that the relatively high incidence of recurrence after the usual nephrectomy for renal carcinoma is due to original extension of the primary growth into the enlarged perirenal veins.

Fig. 6.—Operative site after complete excision of the retroperitoneal tumor, including the anterior and posterior layer of Gerota's fascia.



Anatomically, removal of the perirenal fat with its enlarged venous plexuses, abdominal portions of the ureter and its veins, the ovarian or internal spermatic veins, and the adrenal vein and therefore the adrenal gland on the affected side is recommended as a logical extension of the surgical treatment of these cases (Fig. 6).

Certainly it follows that the transperitoneal approach allows the surgeon to accomplish a more nearly perfect operation with regard to the above technical extensions of the procedure, whether the tumor be small or large.

STATISTICAL FACTORS

Reevaluation of our series [2] with follow-up to January, 1954, revealed a total of 111 retroperitoneal tumors observed: 63 Wilms' tumors and 48 neuroblastomas. Of all the cases (78) observed up to three or more years ago, 24% of the Wilms' tumors are alive and 11% of the neuroblastomas (Table 4).

TABLE 4.—*Reevaluation of Neuroblastoma and Wilms' Tumor* *

111 Retroperitoneal Tumors *	
Wilms' Tumor	Neuroblastoma
63	48
3-Year Survival (All Cases Observed)	
24%	11%

* L. A. Children's Hospital Series, 1928 to 1954 (pathological classification after operation or autopsy).

TABLE 5.—*Reevaluation of Neuroblastoma and Wilms' Tumor*

Diagnosis and Treatment * by One Year of Age (3-Year Survival)	
Wilms' Tumor	Neuroblastoma
36%	30%

* Surgical excision and postoperative roentgen therapy.

TABLE 6.—*Reevaluation of Neuroblastoma and Wilms' Tumor*

Neuroblastoma: "Localized Tumor"
3-Year Survival
55%
(With complete or incomplete excision and postoperative roentgen therapy.)

In the Wilms' cases in which nephrectomy seemed feasible and was attempted, there was a 30% three-year survival rate and the average age of the survivors at operation was 40 months. Those diagnosed and operated by one year of age have a 36% three-year survival rate (Table 5). The neuroblastoma cases diagnosed and treated by one year of age had a 30% three-year survival rate (Table 5). The average age at operation of the survivors was eight months. This is a very significant fact and again emphasized the importance of early diagnosis as some 87% of the neuroblastoma group entered the hospital in a terminal state or with obvious distant metastases.

In the small group in which a localized tumor was found and treatment consisted of complete or incomplete excision with postoperative roentgen therapy, there was a 55% three-year survival rate (Table 6). These statistics show the same trend regarding prognosis in relation to the age of the patient when first treated as those of the outstanding series from Boston Children's Hospital.

A diagnostic aid of special importance is an intravenous pyelogram. This procedure revealed a retroperitoneal mass in 87% of the cases and assured the surgeon of a functioning kidney on the opposite side (Fig. 7).

Wilms' tumor (termed embryomas or adenomyosarcomas of the kidney) may contain muscle, fat, nerve, glands, cartilage, bone, glomeruli, and tubules. With this fact in mind, one can better understand the unpredictable response of Wilms' tumor to irradiation. A careful 10-year review of the literature for statistical evaluation of roentgen therapy [1] revealed no significant difference in results with preoperative or postoperative roentgen therapy alone or in combination (Table 7). Postoperative roentgen therapy is generally accepted in the treatment of Wilms' tumor and especially in the treatment of neuroblastoma.

Fig. 7.—Pyelogram showing an intrinsic left renal mass (Wilms' tumor).

TABLE 7.—*Wilms' Tumor in Childhood: Tabulation from Literature of All Cases Treated by Nephrectomy for Period 1941-1950, with Two or More Years Survival*

	Number of Cases	Survivals	Per Cent
Nephrectomy with pre- and post-operative irradiation	34	10	29.4
Nephrectomy and post-operative irradiation	79	31	39.2
Nephrectomy only	24	10	41.6
Nephrectomy and preoperative irradiation	19	8	42.1
Total	156	59	37.8

COMMENT

It is helpful to keep in mind the clinical concept of the symptoms produced by an expanding retroperitoneal tumor in infants and children. When an abdominal mass is felt, the condition should be considered malignant until proved otherwise. Specific curative treatment of a retroperitoneal tumor, regardless of type, requires surgical intervention.

Reevaluation of the "usual" prognosis given when neuroblastoma is discovered is certainly in order, in view of recent reports of significant series and of particular individual cases. If the neuroblastoma is still confined to the abdomen, then complete or incomplete surgical excision is to be attempted, or at least a biopsy if this is all that can be safely accomplished, followed by judicious x-ray therapy. Because of the radiosensitivity of these tumors and the occasional proven spontaneous maturation, the prognosis may be good in spite of inoperability.

And finally, the transabdominal surgical approach with initial ligation of the renal pedicle and inclusion of Gerota's fascia and the collateral renal veins in the removal of a retroperitoneal tumor involving the kidney is emphasized as essential for an adequate cancer operation. With neuroblastoma or Wilms' tumor, early diagnosis is still the main therapeutic tool, followed by adequate surgery and postoperative roentgen therapy.

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Case Reports

SITUS INVERSUS AND ACUTE CHOLECYSTITIS

Report of a Case

EDWARD R. MUNNELL, M.D.
and
JOSEPH L. PONKA, M.D., Detroit

COGNIZANCE of the relations of the viscera to external markings of surface anatomy is of the utmost importance in the execution of the surgical task at hand. Every surgeon, some time during his practice, will very likely be betrayed by nature and will find that the organ sought after is located in exactly the opposite side from the placement of his incision. He will be dealing with a case of situs inversus.

In spite of the relative rarity of situs inversus, there are reports in the literature concerning its association with surgical diseases. Blegen,[1] in 1949, was able to amass a total of 144 cases which had had operation in the presence of situs inversus. Twenty-four of these had had biliary tract operative procedures. Kullander,[2] in 1950, reported a case of a 63-year-old white woman with recurent episodes of acute cholecystitis in whom cholecystectomy was performed. He emphasized the importance of the x-ray as an aid in the early diagnosis. A case of acute cholecystitis in a patient with situs inversus is presented.

REPORT OF CASE

T. G., a white man of Armenian parentage was originally seen in 1936 at the age of 31 when a diagnosis of infectious arthritis and situs inversus (based on a right-sided heart, liver dullness on the left, and stomach tympany on the right) were made. A biliary drainage in 1937 was reported as suggestive of chronic cholecystitis. During the latter part of 1949, he was hospitalized with a diagnosis of virus pneumonitis. In 1950 the patient was followed in the gastrointestinal clinic because of epigastric pain, bloating, and fatty food intolerance. A gastrointestinal series was reported as showing a normal stomach and duodenum but with complete situs inversus. Cholecystograms were reported as showing nonfunction, but the film was taken in the area of the right hypochondrium. He was placed on a low fat diet.

In August, 1952, he reported to the emergency room, stating that he had had recurrent attacks of epigastric pain, nausea without vomiting, and a more marked intolerance to fatty foods which brought on the symptoms. Examination disclosed epigastric tenderness without guarding or rebound and a temperature of 97.6 F. A flat plate of the abdomen showed the heart on the right, the liver on the left, and a large solitary gallstone lying just below the liver shadow (Figure). The stomach air shadow was on the right. Since he refused admission, further study was not carried out, and he reappeared in November, 1952, with symptoms and findings of acute cholecystitis. The patient did not wish admission for a second time but reappeared 10 days later, stating that the symptoms had persisted while he was at home and had become worse in the past 36 hours. Examination revealed left upper quadrant tenderness, guarding and rebound. Temperature was 99 F., pulse rate was 100 per minute, and the white blood cell count 11,300 with a shift to the left. A repeat flat film of the abdomen again showed situs inversus with a left-sided stone.

The patient was admitted at this time for conservative management. However, six hours after admission the temperature rose to 101 F, pulse to 124 per minute, and the white blood count to 19,400. There was increased tenderness and guarding in the left hypochondrium. He was observed during the night, and the following morning the temperature had fallen to 99.4 F, but the pulse was 130 per minute and the white blood count 27,500. There was now a palpable tender mass in the left upper quadrant which was thought to represent the gall bladder. The patient was prepared for operation.

With the patient under spinal anesthesia, a left subcostal incision was made. When the abdomen was entered, the diagnosis of situs inversus was confirmed by finding a complete reversal of all the abdominal organs. The acutely inflamed gall bladder in the left upper quadrant was blanketed with omentum. This was gently removed by blunt dis-

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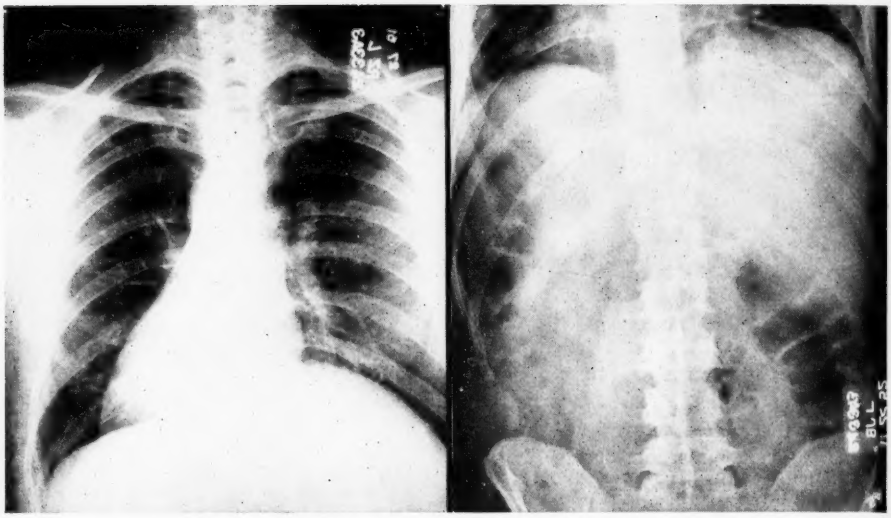
Department of General Surgery, Henry Ford Hospital.

Present address of Dr. Munnell: Veterans Administration Hospital, Oteen, N. C.



Chest film and scout film of abdomen showing situs inversus. Note gallstone in left upper quadrant.

section. It was possible to dissect out and identify the cystic duct and artery and remove the gall bladder without difficulty. A drain was placed in Morison's pouch, and the abdomen was closed in layers. The patient received 1 pt. of whole blood during this procedure. The postoperative course was uneventful except for the development on the second day of mild atelectasis and on the ninth day of urticaria due to penicillin. He was discharged on the tenth postoperative day.

COMMENT

In the case reported the flat plate of the abdomen was of considerable help in ascertaining the true condition of the acute abdominal emergency, since his record was not immediately available when he was seen in the emergency room. Although symptomatically this patient did not have pain directly over the gall bladder, this is not unusual in situs inversus and has been explained on the evidence which suggests that, although the viscera are transposed, the component parts of the nervous system are not.[1]

The records of the hospital have shown situs inversus indexed 22 times in 664,000 registrations. Certainly, some cases have been overlooked, since numerous registrants are emergency or outpatient cases and do not have complete examinations. There are 11 male and 11 female patients in this group. Ten cases were seen from 1926 to 1947 and 12 since 1947. This larger number reported since 1947 is on the basis of the large volume of children seen at this hospital with congenital heart disease. A striking feature in reviewing the cases of situs inversus was the ease with which this condition can be missed by the clinician or radiologist.

SUMMARY

A case of cholecystitis with situs inversus which eventually was treated by cholecystectomy is reported. Greater vigilance on the part of the clinician and radiologist alike is to be sought, since situs inversus can be easily overlooked.

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